Suid-Afrikaanse Tydskrif vir Geneeskunde
South African Medical Journal

Posbus 643, Kaapstad **P.O. Box 643, Cape Town**

Kaapstad, 31 Mei 1958
Weekliks 2s. 6d.

Vol. 32 No. 22

Cape Town, 31 May 1958
Weekly 2s. 6d.

THALASSAEMIA-HAEMOGLOBIN E DISEASE IN A CAPE COLOURED FAMILY

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In 1952 Budtz-Olsen and Woolf[1] described a Cape Coloured family whom they thought to be suffering from thalassaemia (Cooley's anaemia). With the knowledge then available the authors found it difficult to give a genetically satisfactory explanation of the varying severity of the disease in different members of the family. Blood from members of this family has now been re-examined with the help of paper electrophoresis, and it has been shown that the more severely affected children are suffering from thalassaemia-haemoglobin E disease. This was first described in 1954 by Chernoff *et al.*[2] and later by Sturgeon *et al.*[3]

MATERIAL

Fig. 1 shows the pedigree, with the electrophoretic patterns added. The mother, No. 1, was of Cape Coloured stock. This is a mixed people descended from Europeans, Hottentots, Bushmen and Asians, with very little negro admixture. Her mother, since dead, was examined by Budtz-Olsen and Woolf and correctly regared as having thalassaemia minor. The father of the family (No. 2) was a Cape Malay; these Malays are descended from Eastern slaves, not necessarily from Malaya. He died of heart disease some years ago, but it is clear from the findings that he must have been the bearer of the haemoglobin E trait; his relations could not be traced. Blood was taken from No. 3, the eldest child, only after she had received a large transfusion, which added haemoglobin A to the electrophoretic pattern.

Electrophoresis of haemoglobin was carried out as described by Brain.[4] The presence of alkali-resistant (F) haemoglobin was confirmed by denaturation (Singer *et al.*[5]). The other findings are from the paper of Budtz-Olsen and Woolf. The identity of the haemoglobin E was confirmed by Dr. H. Lehmann of St. Bartholomew's Hospital, and by the late Dr. Karl Singer of Chicago.

RESULTS

Fig. 1 and Table I show the pertinent findings, and illustrations will be found in the paper of Budtz-Olsen and Woolf.[1]

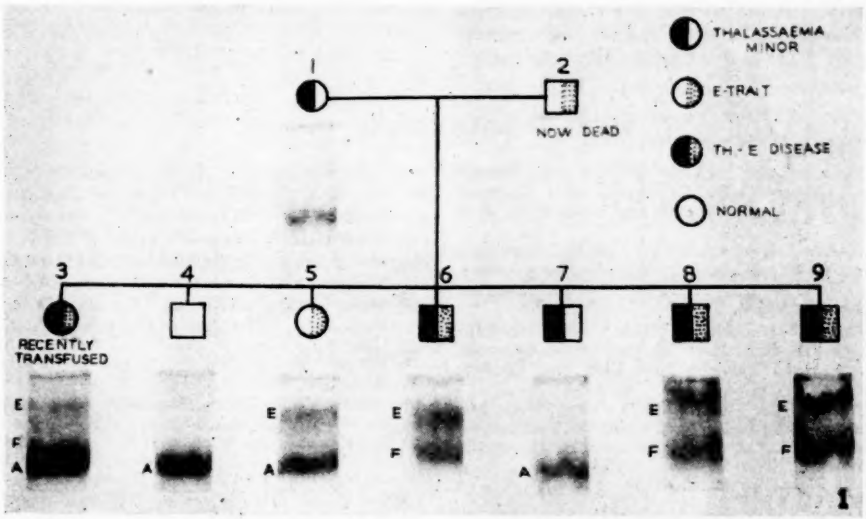

Fig. 1. Pedigree with electrophoretic patterns. The haemoglobin has been applied at the top of each strip and migration has taken place downwards towards the anode.

In this family, a mating between a woman with thalassaemia minor and a man with the haemoglobin E trait has produced in the 7 children every possible genetic combination—normal (No. 4), thalassaemia minor (No. 7), E-trait (No. 5), and thalassaemia-haemoglobin E disease (Nos. 3, 6, 8 and 9). The clinical manifestations of thalassaemia-haemoglobin E disease are much the same in the 4 cases, and agree closely with those described by Chernoff *et al.*[2] All 4 were of stunted growth with mongoloid features. The spleens were enlarged and hard, in one case (No. 3) reaching down to the anterior superior iliac spine. None showed obvious jaundice, but urobilin was occasionally found in the urine of No. 3. This girl, who in 1954 was 24 years old and had needed transfusions 2-3 times a year for the preceding 4 years, also had a persistent granulating ulcer over the lower part of the left tibia. X-ray examination showed generalized osteoporosis in the three subjects examined, but none had the 'hair-on-end' appearance of the skull. The picture of the peripheral blood was indistinguishable from that in thalassaemia major. The bone marrow of No. 3 was examined and showed a considerable increase in erythropoiesis with a preponderence of normoblasts. All the subjects were negative for sickling.

TABLE I. FINDINGS IN THE AFFECTED FAMILY

Subject No.	*Haemoglobin g.%*	*Hypochromia*	*Microcytosis*	*Anisocytosis*	*Target cells*	*Nucleated red cells*	*Reduced osmotic fragility*	*X-ray signs*	*Haemoglobins on electrophoresis*	*Diagnosis*
1.	11·8	+	+	+	+	—	+		A	Thalassaemia minor
3.	2·8	++	++	+++	++	++	+	+	EFA	Th.–Hb.E disease after transfusion
4.	12·2	—	—	—	—	—			A	Normal
5.	13·9	—	—	—	—	—			AE	E-trait
6.	9·3	+	+	++	++	+		+	EF	Th.–Hb.E disease
7.	9·6	—	+	++	+	—		+	A	Thalassaemia minor
8.	5·9	++	+	++	++	++			EF	Th.–Hb.E disease
9.	7·8	++	+	++	++	++			EF	Th.–Hb.E disease

Thalassaemia-haemoglobin E disease is thus clinically and haematologically (unless the haemoglobin is examined) indistinguishable from thalassaemia major, though perhaps rather less severe. Whereas in thalassaemia major the haemoglobin consists principally of F (foetal) with some A (adult) and no E, in thalassaemia-haemoglobin E disease it is made up of E and F components. The difference in electrophoretic mobility between haemoglobins F and A is well shown in the adjacent strips 5 and 6, representing the symptomless E-trait (haemoglobins E and A) and thalassaemia-haemoglobin E disease (E and F).

DISCUSSION

Haemoglobin E is relatively common in some oriental peoples, such as Thailanders,[2] Burmese[6] and Indonesians.[7] Thalassaemia also is common, at least in Thailand. The Coloured population of Cape Town contains a considerable oriental 'Malay' element, and the father of the family here described was in fact of 'Malay' extraction. Haemoglobin E, however, is not often found among the Coloured population. No example of it was seen in an electrophoretic study of 430 subjects, including 'Malays' (Brain[4] with additions). Thalassaemia, in its major form at any rate, is very rare in this population. Thalassaemia-haemoglobin E disease is therefore certainly very uncommon in South Africa.

Although in Fig. 1, for the convenience of drawing, the genes for thalassaemia and for haemoglobin E are shown as if allelomorphs, this is not so.[8, 9] The subjects with thalassaemia-haemoglobin E disease described here are in fact double heterozygotes. The combination of thalassaemia with homozygous haemoglobin E has recently been described from Thailand.[9]

We are grateful to Drs. S. Friedlander, D. M. Krikler and C. P. Relief for assistance. Dr. H. Lehmann and the late Dr. Karl Singer kindly examined the haemoglobin of case 3. One of us (P.B.) received a grant from the Dr. C. L. Herman Fund of the University of Cape Town.

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Suid-Afrikaanse Tydskrif vir Geneeskunde: South African Medical Journal

VAN DIE REDAKSIE

SPANNING VAN DIE MAAGMOND

Onlangse studies het veel aan die dag gebring aangaande die aard van die funksieversaking by die toestand bekend as kramp of spanning van die maagmond, maar voordat die verslae bestudeer word, moet ons eers die huidige menings insake slukdermsluitspiere uiteensit. Nadat dit lank die onderwerp van meningsverskil was, is dit definitief bewys dat daar 'n intrinsieke sluitspier onder in die slukderm is, omtrent 2-4 cm. bokant die maagmond; dit is bewys deur die meet van drukverval oor die streek waar die slukderm by die maag aansluit.[1] Die normale slukderm besit twee sulke streke van hoë druk—daar is nog een aan die bo-ent; en hierdie twee streke verhoed respektiewelik dat die maaginhoud en lug in die ingewand versluk word.[2] Dié deel van die esofaag tussen die onderste sluitspier en die maagmond is van groot belang by maagmondkramp; Lerche (aangehaal deur Gould en Bernhard[3]) noem dit die ‚voorhof'.

By maagmondkramp word die voorhof vernou, en daar is 'n wisselende, maar gewoonlik aansienlike, vergroting van die liggaam. Dit was eers gemeen dat die vernouing deur kramp veroorsaak was, maar hierdie verduideliking was maklik verwerp; die vernoude streek bied geen weerstand teen die passeer van stawe nie. Dit is dan as 'n groot stap vorentoe beskou toe Hurst en Rake,[4] in die loop van studies van outopsie-materiaal van gevalle van maagmondkramp, gevind het dat daar 'n vermindering in die aantal senuweeknoopselle in die vernoude deel was. Met dié feit as grondslag het hulle die teorie geopper dat die mondgedeelte van die slukderm nie kon ontspan nie (maagmondkramp). Hierdie teorie is egter nie sonder meer aanvaar nie; en dit blyk dat onlangse navorsing aan die Guy-hospitaal waar Hurst voormalig werksaam was, hierdie stelling omvergewerp het.[2] Gedurende operasies vir verskillende slukdermafwykings, o.a. maagmondkramp, is spierstrokies verkry uit die voorhof; wat gevalle van maagmondkramp sowel as beheergevalle betref het, kon doeltreffende aantalle senuweeknoopselle gewoonlik histologies gedemonstreer word, en biochemiese toetse het bewys dat hierdie selle wel bekwaam was. Trounce *et al.*[2] vestig die aandag daarop dat ná afsterwe die voorhof nie so duidelik uitkenbaar is nie, en stel voor dat die materiaal van Hurst se gevalle miskien in werklikheid uit die vergrootte gedeelte afkomstig was. Volgens hulle is die liggaam van die slukderm abnormaal by gevalle van maagmondkramp en kan daar 'n gebrek aan senuweeknoopselle wees. Dit is nie duidelik wat hierdie vermindering veroorsaak nie, maar dit is *nie* te wyte aan sekondêre inflammasieveranderings nie.

Die werk van Kramer en Ingelfinger[5] lewer verdere bewyse van 'n verspreide stoornis in die senuweevoorsiening en beweeglikheid van die slukderm by gevalle van maagmondkramp; hulle het naamlik 'n abnormale reaksie op cholinergiese prikkeling aangetoon. Anders as normale mense of lyers aan ander slukdermafwykings, ondervind pasiënte met maagmondkramp geweldige en dikwels pynlike sametrekkings van die slukderm ná 'n spierinspuiting van 3 -6 mg. metacholien (Mecholyl). Hierdie krampe kan deurligtings-

EDITORIAL

ACHALASIA OF THE CARDIA

Recent studies have shed much light on the nature of the dysfunction in the condition known as achalasia of the cardia or cardiospasm, but their consideration should follow a brief statement of current views about oesophageal sphincters. After prolonged controversy, firm evidence of the existence of an intrinsic lower oesophageal sphincter about 2–4 cm. above the cardia has been provided by the measurement of pressure gradients across the region of the gastro-oesophageal junction.[1] The normal oesophagus possesses two of these high-pressure zones, there being another at the upper end; these respectively prevent the aspiration of stomach contents and air into the viscus.[2] The part of the oesophagus between the inferior sphincter and the cardia is of great importance in achalasia; it has been called the 'vestibule' by Lerche (quoted by Gould and Barnhard[3]).

In achalasia there is constriction of the vestibule surmounted by variable, but usually considerable, dilatation of the body. At first the narrowing was attributed to spasm, but this explanation was easily disproved; the contracted area offers no resistance to the passage of bougies. It then appeared to be a great advance when Hurst and Rake,[4] using material obtained at autopsy from cases of achalasia, stated that there was a diminution in the number of ganglion cells in the narrowed part. On this basis they postulated that there was a failure of relaxation (achalasia) of the cardiac portion of the oesophagus. However, this thesis did not achieve unquestioned acceptance; and recent work from Hurst's old hospital, Guy's, appears to have disproved it.[2] At operations for various oesophageal disorders including achalasia, strips of muscle were obtained from the vestibule; in both achalasia and the control cases adequate numbers of ganglion cells could usually be demonstrated by histological means, and biochemical tests showed that these cells were functioning. Trounce *et al.*[2] point out that after death the vestibule may become less obvious, and suggest that in Hurst's cases the material may really have come from the dilated part. According to them, the body of the oesophagus is abnormal in achalasia and may be deficient in ganglion cells. The reason for this diminution is not known, but it is not due to secondary inflammatory changes.

Further evidence of a widespread disorder of oesophageal innervation and motility in achalasia comes from the studies of Kramer and Ingelfinger,[5] who have demonstrated an abnormal response to cholinergic stimulation. Unlike normal people or patients with other oesophageal disorders, sufferers from achalasia experience violent and often painful contractions of the oesophagus after an intramuscular injection of 3–6 mg. of methacholine (Mecholyl). This can be observed fluoroscopically and is held to be of use in the diagnosis of

gewys waargeneem word en dit word beweer dat dit nuttig is by die diagnose van maagmondkramp. Daar word voorgestel dat hierdie spieraksie 'n voorbeeld van Cannon se wet is, nl. dat 'n orgaan wat gebrekkig is aan senuwee buitengewoon gevoelig is vir sekere chemiese prikkels.[6]

By maagmondkramp is die funksionele afwyking blykbaar dat daar by die slukbeweging nie 'n ware peristaltiese golf ontwikkel en langs die slukderm af beweeg nie; gevolglik ontvang die slukderm nie sy normale prikkeling om te ontspan nie. Daar is dus ‚spanning' by die maagmond, maar die gebrek aan ontspanning is ondergeskik aan verspreide siekte van die liggaam van die slukderm.

Daar is vandag nog geen definitiewe behandeling nie. Hoewel die nitriete soms nuttig is, help ander kramp-teenmiddels nie en anticholinergiese middels kan erger dan nutteloos wees; 'n spierinspuiting van 25-50 mg. methantelien bromied (Banthine) by normale gevalle kan 'n verbygaande toestand, wat na maagmondkramp lyk, in die hand werk.[7] Hierdie feit ondersteun die idee van senuweegebrek by die etiologie van hierdie siekte. Dit kan by baie gevalle nodig wees om die spiervesels van die voorhof te skeur sodat die voedsel deur swaartekrag, en met die hulp van sametrekkings van die keelholte en die boonste gedeelte van die slukderm, in die maag kan kom. Dit kan gewoonlik bewerkstellig word deur skerp rekking met 'n hidrostatiese sakkie onder X-straal-beheer; indien hierdie metode nie slaag nie, of as die slukderm té verrek en kronkelend is om die sakkie sekuur in posisie te plaas, is snykundige ingreep moontlik nodig (Heller se operasie). Dit beantwoord gewoonlik die doel, maar as die verwyding té verregaande is, kan maag-slukdermterugvloeiing plaasvind, met gevolglike peptiese slukdermontsteking.

Hoewel die behandeling van maagmondkramp dus maar 'n moeilike saak bly, is daar nietemin vooruitgang, en al is die etiologie nog duister, is die patogenese tog al bekend. Maar daar moet nog baie aan hierdie probleem gewerk word voor ons aanspraak daarop kan maak dat hierdie seldsame maar belemmerende siekte oorwin is.

achalasia. It is suggested that this behaviour is an example of Cannon's law, that a denervated structure is abnormally sensitive to certain chemical stimuli.[6]

In achalasia the dysfunction appears to be that with swallowing a true peristaltic wave does not develop and move down the oesophagus; consequently the vestibule does not receive its normal stimulus to relax. There is thus 'achalasia' at the cardia, but the failure to relax is secondary to diffuse disease of the body of the oesophagus.

At present there is no definitive therapy. Although nitrites are occasionally of some use, other antispasmodics do not help, and anticholinergic drugs may be worse than useless; the intramuscular administration of 25–50 mg. of methantheline bromide (Banthine) to normal subjects may induce a transient state resembling achalasia.[7] This is another point supporting the denervation concept of the aetiology of this disease. Many cases will need to have the muscle fibres of the vestibule ruptured, so that food can enter the stomach by gravity and with the aid of pharyngeal and upper oesophageal contractions. This can usually be accomplished by brusque dilatation with a hydrostatic bag under fluoroscopic control; if this fails, or if the oesophagus is too dilated and tortuous to permit of accurate placement of the bag, operative section (Heller's operation) may be required. This usually works, but if the dilatation is too extensive, gastro-oesophageal regurgitation may result, with consequent peptic oesophagitis.

Thus, while the treatment of achalasia remains difficult, there have been advances, and the pathogenesis, if not the aetiology, is now known. But much further work is required before we can say that this uncommon but disabling malady has been mastered.

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ABNORMAL VAGINAL DISCHARGE

A PRELIMINARY REPORT ON THE WORK DONE BY THE 'VAGINAL DISCHARGE CLINIC' OF THE GROOTE SCHUUR HOSPITAL, CAPE TOWN*

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The natural moisture of the vagina varies in quantity and quality according to the age, menstrual phase and gestational stage of womankind. This natural moisture may be increased during periods of stress and strain and, under these conditions, may extrude through the vulva. This may or may not produce slight discomfort, which is more psychological than physical in nature. This type of discharge has rightly been referred to as 'the thermometer of woman's health'. On the other hand, should the vaginal discharge persistently or paroxysmally be copious, it becomes the source of physical and sometimes gross mental discomfort. This unpleasantness may be increased unless adequate steps are immediately instituted for complete aetiological investigation, followed by thorough institution of treatment. Investigations directed at exposing the aetiological agent are time-consuming and may not be adequately undertaken in a busy gynaecology outpatient department. Furthermore, these discharges may but be the very early symptoms and signs of graver diseases. For these reasons, i.e. primarily for the patient's benefit and, secondarily, for stimulus of thought, a vaginal-discharge clinic was started in this hospital. In addition, in conjunction with the erosion and endocrine clinics, this investigation forms part of a wider drive aimed at the discovery or prevention of genital cancer.[1]

Several authors call attention to the fact that trichomonas infection is a disturbing factor in the study of vaginal smears, and may result in false interpretations.[2, 3] Davis[1] has recently tried to correlate his findings that trichomonas infection was

*A paper submitted to the South African Medical Congress, Durban, September, 1957

3½ times more frequent among hospital patients than among private patients, as were the reports of malignant cells in the smears. He concluded that possibly the fact that the uncircumcised male may carry trichomonads under the prepuce might be a reason for the increased incidence of carcinoma. Authorities differ on the value of these special clinics. Lewis[5] in his recent book states that, although they offer excellent opportunity for postgraduate teaching and the follow up of a large number of cases, he doubts whether the best interests of the patient are served. Donald[6] says a special clinic can investigate these patients far better than a busy out-patient department; all the necessary equipment is at hand, more efficient organization is possible, and better facilities are offered for research.

Often on examination, although the outstanding complaint was a vaginal discharge, no discharge could be detected, or not enough for investigation, even though patients were warned not to bath or douche beforehand. Even on examination at a later date very little discharge could be found and undoubtedly in some of these cases a cancerphobia or syphilophobia existed. This type of individual is reassured and her fears allayed by the thorough examination. Another difficulty was the very high incidence of defaulters (47%). It was a comfort, however, to note this difficulty in other reports.

Great difficulty was encountered in persuading husbands to report for examination. This aspect has been neglected but it is intended to investigate it adequately in future. Many patients did not want to discuss the matter with their husbands. Husbands, who were free from symptoms, were not keen on leaving their work in order to visit the hospital. Another insurmountable difficulty encountered was that a number of the patients were unmarried.

Method of Investigation. A drop of the discharge was examined directly in saline, and a swab taken for culture—obviously water was the only instrumental lubricant used. Since June vaginal and cervical smears have also been taken for examination according to the method described by Papanicaloau. It was a source of encouragement to find that many Papanicaloau smears were found positive for trichomonads, and sometimes monilia when the direct examination failed to reveal these organisms. The smear technique seems to be more reliable than the direct examination for trichomonad infections. The incidence of trichomonas infection is higher in this than in other reports (Table I). In all probability

TABLE I. INCIDENCE OF DIFFERENT CAUSES

	Donald[6]	*Ray and Maugham*[26]	*Groote Schuur*
Trichomonas	37·4	39·6	49·2
Monilia	16·2	13·6	24·5
Trichomonas and Monilia	7·7	—	4·6
Non-specific	33·5	44·2 (Haemophilus)	21·7
Extras	5·1	2·6	—

it would have been higher still if we had used this technique from the beginning. Other authorities have reported that smears show a very high percentage of trichomonads. The culture has not been used for recovering trichomonads; however, its use has been stressed by Kupferberg,[8] Sorel[9] and Whittington.[13]

Treatment. Once a firm diagnosis has been made, treatment as a rule follows set patterns. Difficulties are however often encountered, the cause of which have to be found, and appropriate counter-measures taken.

Trichomonal Infection. The literature on this subject is voluminous and countless procedures have been advocated. Most treatments, even simple douching, will usually afford temporary relief, and may possibly cure the patient. Some patients, apparently cured, abandon treatment, which is one of the reasons for the high rate of recurrence.

Treatment has to be as simple as possible. S.V.C. or Floraquin pessaries were used daily for 8 weeks, the menstrual period included. For the next few months, pessaries were used for a few days after the period, i.e. when relapse tends to occur. In some resistant cases, oestrogynedron was tried with success—a cream containing oestrogen, sulphonamide and lactose in a vehicle which disperses well in the vagina.

TABLE II. INCIDENCE OF TRICHOMONAS IN THE MALE

Donald[6]	Husbands examined in 4 of the relapsing cases and no positives found
Barnes *et al.*[12] ..	8 husbands examined, 2 positive
Whittington[13] ..	27% of husbands whose wives infected
Coutts *et al.*[14] ..	40% of husbands whose wives infected
Senecca and Ides[15] ..	16% of 926 symptomless recruits
Feo[16]	15·5% of symptomless males
Perl *et al.*[17]	2·7% only when urine examined. 58% in semen culture.

Good results with this therapy have been claimed by Briegleb.[9] However, until the method of reinfection is clearly understood, and we have a systemic form of treatment as well, the results will continue to be disappointing. Authorities differ on the role played by the male. Baird[10] states that the treatment of the male up to now has not proved of much practical importance, and Donald[6] that examination of the

TABLE III. USE OF ORAL TRICHOMONACIDE IN FEMALE. (TRITHEON AMINITOZOLE TRICHORAD)

Barnes *et al.*[12]	Used with Acijel	6 out of 37 patients cured (16%)
Plentl *et al.*[18]	Tritheon alone	38% cured, 67% asymptomatic
Perl *et al.*[17]	Tritheon with Acijel	33% cured (without treating husband)
Catterall and Nicol[19]	(*a*) Tritheon alone	100% failure in 20 cases
	(*b*) Tritheon and Penotrone pessaries	9 failures in 10 cases, 1 defaulter
Cuthbert and Husband[20]	Both male and female treated	4 successes and 2 partial successes in 41 cases
Gardner and Dukes[21]	44 patients treated	All positive within 13 days of completion on treatment although half showed clinical improvement
Groote Schuur Hospital	54 cases out of 87 cured without Tritheon (62%)	
	29 cases out of 52 cured with Tritheon in addition (56%)	

male in relapsing cases did disclose the presence of trichomonas. Table II shows that trichomonad infection in the male is becoming increasingly recognized. We have been unable to examine the males so far, but have advised that the male should use a condom for at least 3 months. Recent work on oral treatment of vaginal discharge with trichomonacide-aminitrozole (Tritheon trichorad) has raised great hopes, but our results like those of others, have been disappointing. Table III shows results obtained at Groote Schuur Hospital and by others elsewhere. There may be a place for its use in the male, and we are investigating this side further. (Table IV)

Caterall and Nicol[19] make a plea that new drugs should not be put on the market and advertised until there has been a really adequate clinical trial. Table V shows the results

TABLE IV. USE OF ORAL TRICHOMONACIDE IN MALE

Perl *et al.*[17]	26 men treated: (1) 16 cured after 1 course, (2) 2 cured after 2 courses, (3) 10 defaulters
Barnes et al.[12]	8 husbands examined: 2 were positive and successfully treated
Catterall and Nicol[19] ..	6 males treated and Trichomonas found in the urine of all 6 afterwards
Groote Schuur Hospital	5 males treated and Trichomonas still found in urine of 3 after the treatment

obtained by Caterall and Nicol with an antibiotic, trichomycin, isolated in Japan by Hosoya *et al* and stated by them to be effective against trichomonads.[22] Caterall and Nicol do not confirm these claims, either with systemic or local use.

TABLE V. ORAL USE OF TRICHOMYCIN (CATERALL AND NICHOL[19])

	No. Treated	*Cures*	*Failures*	*Defaulters*
Trichomycin oral ..	44	0	41	3
Trichomycin pessaries..	23	1	17	5
Acetarsol pessaries ..	23	9	8	6

Monilial Infection. Treatment with gentian violet has been replaced by the insertion of Mycostatin pessaries. Up to the present there have not been many reports published on this therapy. Stallworthy[23] reported that he had extremely good results. Jennison and Jones[24] report 47 cases free from infection in 1 week out of 53 cases treated with Mycostatin (88%) as compared with 17 cures out of 36 treated with gentian violet (42%). Of 18 cases not responding to gentian violet, 16 were cured with Mycostatin. The relapse rate after 4 weeks was 46% with gentian violet as compared with 21% with Mycostatin. Jennison and Jones state in a letter that, since their article, they have improved their results by using the pessaries for 14 days. We have found that the pessaries should be used for at least 3 weeks. It has been suggested that drug resistance may develop,[25] but this has not been established.

At Groote Schuur Hospital we treated 70 cases of monilia infection with Mycostatin pessaries. Of these, 44 defaulted but the 26 patients who reported were all cured—and probably most of the defaulters as well, for otherwise the unpleasant pruritis that is associated with monilial infection would have brought them back.

It has been maintained that monilial infection never occurs unless there is at any rate intermittent glycosuria, but this is not so. The use of antibiotics has led to a marked increase of monilial infection, and it has been suggested that it is wiser to give Mycostatin by mouth as well when using broad-spectrum antibiotics, and in pregnant or diabetic patients it is probably wise to do so. Stone and Mersheimer[26] showed in a controlled series that no case developed vaginal moniliasis while receiving the combination, whereas the reverse was obtained when Mycostatin was not included in the therapy.

Non-specific Vaginitis

The so-called non-specific vaginitis comprises a large number of cases. It is defined as a diverse group of vaginal infections which cannot be attributed to any specific pathogenic organism. These cases are associated with mixed bacterial flora. In some of them associated ovarian disturbances are found. In the extreme case we have the so-called atrophic vaginitis; others are secondary to cervical disease, but in the vast majority no specific cause has been found. In 1955 Gardner and Dukes[22] reported the presence of a specific organism, named by them *Haemophilus vaginalis*, which they thought to be the aetiological agent in the majority of cases of non-specific vaginitis. It was cultured from 127 out of 137 cases. The organism isolated fulfilled Koch's postulates for pathogenicity, i.e. they established the disease in 11 out of 15 volunteers. The organism was also recovered from 45 out of 47 of the husbands in Gardner and Dukes' series. Ray and Maugham[28] also report that this organism was responsible for the majority of their cases. They say, however, that they experienced considerable difficulty in culturing the organism, and that they were unable to duplicate Gardner and Dukes' culture records. We have also experienced great difficulty in culturing this organism. In our 90 cases of 'non-specific' vaginitis there were 8 cases in which the gram stain and wet preparation showed 'clue cells' and large numbers of small negative rods. In 3 of these cases we have isolated on culture an organism which appears to have all the characteristics of *Haemophilus vaginalis*.

Gardner and Dukes[27] record 29 cures out of 60 cases of haemophilus vaginitis treated with 'triple sulfa cream' (they gave up the tetracycline treatment of these cases because of the development of monilia. They cured 29 out of 30 infected husbands with tetracycline. Ray and Maugham[28] obtained 27 cures (75%) out of 36 cases of haemophilus vaginitis by treating with 'triple sulfa cream'. There were 9 failures and 4 of these they cured with Mysteclin (Mycostatin plus tetracycline) with no development of monilial infection. All the infected husbands they treated with Mysteclin were cured.

Recently new broadspectrum antibacterial chemical compounds have made their appearance. Claims have been made regarding their effectiveness in trichomonas, monilial and haemophilus vaginitis. These claims have not been substantiated in our clinic against trichomonas vaginitis and monilial vaginitis. However, work is proceeding to evaluate their usefullness in the haemophilus cases. Table VI shows that Gardner and Dukes[19] have found Hexetidine (sterisil) of little value in trichomonas and monilial vaginitis. On the other hand, they obtained with it 83% of cures in haemophilus vaginitis, and Ray and Maugham[28] report success with it in 5 out of 6 cases of haemophilus vaginitis.

TABLE VI. HEXETIDINE (STERISIL) IN THE TREATMENT OF VAGINITIS (GARDNER AND DUKES)

Condition	*Patients Treated*	*Cures*	*Failures*	*Cured %*
Haemophilus vaginalis Vaginitis	79	66	13	83·5
Trichomonas Vaginitis	42	0	42	0
Vaginal Monilia ..	34	4	30	11·8

Our results at Groote Schuur in so-called non-specific vaginitis have so far been disappointing. In 61 cases, after deducting 22 defaulters, 19 have been cured and 20 were not cured when last seen. However, we are now concentrating on these cases and hope in the future to publish a paper on this subject. We have recently also been more successful in culturing the Haemophilus.

CONCLUSION

In this preliminary report, these 3 main causes of disturbing vaginal discharge have been briefly discussed. The more detailed analyses will be left for a later day. It cannot be sufficiently emphasized that to be rid of an ever-present vulval moistness, and the mental anguish associated with an odour that may be emitted by this discharge, is a gift

every patient is most grateful to receive. Simple albeit prolonged treatment based upon accurate aetiological assessment almost invariably assures this happy result.

SUMMARY

1. The reasons for starting a vaginal discharge clinic are discussed, and the advantages and disadvantages as well as the difficulties encountered.

2. The methods of investigation employed are described, and the value of the Papanicaloau smear noted.

3. The treatment of the different types of vaginitis is discussed with particular reference to (a) the value of the new systemic trichomonacide Tritheon, (b) Mycostatin (Nystatin) in monilial vaginitis, and (c) the treatment of 'non-specific' vaginitis and the recently described haemophilus vaginitis.

4. Results of treatment are compared with other published series.

I should like to thank Prof. James T. Louw for his constant guidance, encouragement and help, Dr. Theo. Sacks and Mr. N. D. Constantine for their cooperation in the laboratory investigations, and Sister T. Fox for organizing the clinic so efficiently.

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DERMATOLOGY OF THE INFANT*

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Children under 3 comprise 9% of the cases in my private practice. Infancy, of course, is supposed to end at 2 years, but skin diseases do not always respect this boundary. Consequently I have extended it to 3 so as to broaden the perspective of my survey. Taking 2,000 consecutive White patients referred to me, 3.5% were under 1 year, 3% between 1 and 2 years, and 2.5% between 2 and 3. Four disease groups together make up roughly 70% of these cases, viz. infantile eczema together with infantile seborrhoeic dermatitis, 42%; papular urticaria, 14%; scabies, 6.5%; and pyodermas, 6.5%.

Dermatologists tend to be favoured with a certain type of infant patient and, in what follows, this selective emphasis will be apparent. My own offerings on this subject represent little more than one man's view of the dermatoses of White infants seen in consultation in Pretoria. It is hoped that they will make it simpler for those with other experiences to assess their observations from other places and populations within the Union.

ECZEMA

Eczema is the dermatosis of infancy *par excellence*. In my cases of eczema in children below the age of 3, only 10% were between 2 and 3, while 90% were under 2. Most authors apparently find it easier to classify the cases than I do. The clinical variations of eczema in infancy are almost as complicated as in adults. In most cases, one merely has to decide if the baby has infantile eczema or seborrhoeic dermatitis, and this can be done by referring to a table of comparative criteria. This is at any rate what one is often led to believe. Almost everything from 'acidity' (*suur*) rashes on the cheeks to severe generalized dermatitis must be brought home to one of these alternatives. In between these two extremes one may consider sudden morbilliform rashes when the baby touches the cat, 'sweat' and 'heat' rashes, eczematous reactions to non-specific traumata, scattered eczemas of a type seen in adults, fixed impetiginous eruptions, acute follicular exanthemata, etc., which would not readily be related to infantile eczema but for their occurrence in infants. Accredited treatments are similarly confusing to my mind. Paediatricians may refer confidently to vipenta drops, noctec, daxalan, kolpix D, happy-tappy crackers and so forth without always creating a clear impression of why and wherefore.

About a third of my cases refuse to be classified with ease. Here are some clinical features of these problem cases:

(*a*) A number of cases under this heading are mentioned in the sections on follicular disorders and impetigo.

(*b*) Lesions in the elbow and knee flexures are the starting point of a variety of types of disorder. They may start early or late in infancy. They may or may not itch, may be glazed or dry, smooth or lichenified, annular or flame-shaped, vaguely or sharply outlined, and may disseminate elsewhere with annular or poral eczematides, with outlying papules having a resemblance to insect bites. Anaemic haloes may surround many of the papules.

(*c*) It is commoner for seborrhoeic dermatitis to metamorphose into infantile eczema than for infantile eczema to develop features of an eczematide. Theories depending on coincidence to explain these interrelationships are rather strained.

(*d*) Cases of typical seborrhoeic dermatitis may show otherwise a perfect atopic constellation of associated features.

(*e*) As in adults, so may infants present a type of fixed eruption in seborrhoeic dermatitis.

(*f*) Scattered patchy eczemas are not rare, and may be virtually confined to the limbs. They may either lichenify or change to the picture of pityriasis alba.

* A paper submitted to the South African Medical Congress, Durban, September 1957.

The next vexation comes from the explanations offered for these findings. However benign, modest or gracious a writer may be, it is still misleading to put in the foreground classifications that depend on things that the clinician cannot put to the test. A soothing dispenser of dogma is actually no trouble to take apart, but one may be diverted by false emphasis into thinking that many statements are secure which have been accepted without adequate support. Allow me to paraphrase some of the kinds of statement here which to my mind are open to objection.

Here is one about seborrhoeic dermatitis which bristles with unproven hypotheses: 'Seborrhoeic dermatitis is not primarily allergic, but may occur in allergic infants. The babies with seborrhoeic dermatitis are greedy feeders and eat more than the skin can handle, which causes aggravation of the rash.'

Here is another about atopic dermatitis which embodies some irrational ideas: 'Atopic dermatitis is an immunological disorder, in which immunological treatment attempts to get at the cause. Failures are due to the intervention of an additional X factor, whose intrusion does not invalidate the basic mechanism. The immunological mechanisms involved often require both higher quantities of the allergen and longer times for action than may normally be expected, but the original sensitizing substances may depend on what the mother was exposed to in pregnancy, or what the animal ate whose food products are eaten by the patient. Removing the offending substance is often followed only gradually by clinical improvement. Wool acts as an allergen by inhalation, picking out the weak spots in the skin. It also acts as a contact sensitiser. Itching is a constant symptom.'

My own treatments differ in no way from the empirically established methods, and there is little known on the subject that has not been adequately discussed by many writers. As others have done in the past, we are at present thinking of alterations in the ionic environment of the skin as a possible guide to treatment.

LICHEN URTICATUS (STROPHULUS; PAPULAR URTICARIA)

In Pretoria this is a disease which starts in the summer months, from November to February. I have good records of two summers, and the highest peak was reached in January, 1956. I have seen hardly any cases which started between April and October. The ages of my patients ranged evenly between 6 and 32 months, indicating that the age of the infant was of negligible importance compared with the time of year. Lichen urticatus is not a disease limited to infancy, and the cases occurring in childhood include patients with bullous prurigo, which lie outside our survey.

Regarding the cause, I have found that lichen urticatus of atopic origin cannot account for more than 5% of all lichen urticatus cases I have seen in infants. The parents have sometimes blamed water-melon, grapes, tomatoes, apricots and ice cream as causes. Now diet in lichen urticatus is a topic on which one may separate medical writers into the tender-minded and the tough-minded, just as one can do with attitudes on other subjects. The tender-minded will say: 'Let us try to get this baby well soon, and even if raw fruit is not the only cause, it will be wise to withhold it.' The tough-minded will think: 'Let us give this baby all the food and fruit that the parents think are bad for it and see what happens. If the rash fails to come out, the sooner we stop being hedged about by nonsense the better.' The tough-minded have seemingly won the day and have shown, despite the most suggestive evidence to the contrary, that lichen urticatus is unconnected with atopy or any article of diet. As one of the tough-minded has told me: 'Either grandmothers derive their observations from reading dermatological text-books or, if they don't, then the text-books are written by those with the mentality of grandmothers.'

Looking over my own cases there are several points which have persuaded me indirectly of the importance of insect bites in the aetiology. The seasonal factor has been noted already. I have seen several cases brought on by severe onslaughts from mosquitoes or fleas such as may happen if a mosquito net is left off for a night or if the family goes for a holiday to a flea-ridden farm or to the coast. It should be noted, though, that according to my observations, lichen urticatus may start more than a week after the first exposure to a strange bloodsucking population. Parents who hastily deny that the trouble is related to bites can seldom tell one how the baby's skin reacts to an actual mosquito bite. However, to prevent an insect sucking blood, one merely has to bind up a limb with elastoplast, and I have found that lichen urticatus heals and remains clear on the limb so protected. It is so simple that I would like more people to try it on their cases. In a few of my cases an anti-diphtheria inoculation seemed to bring on an attack of lichen urticatus, but the observations are too scanty to warrant conclusions.

For prophylaxis and treatment during infancy, one can usually insist on a mosquito net, though when one is outside the malaria belt, it is harder to get people to sleep under nets. Mosquito repellents, such as dimethylphthalate (Mylol) may be used, but do not always work, and some mothers object when the solution takes the paint off their finger nails. Besides these measures, I have been using a watery shake lotion with 2% phenol and 5% DDT to be applied twice daily in a thin layer to the whole skin surface. This is aimed at fleas and the itching. If the regime is strictly followed, there are seldom any new spots over the 10 days required for the old spots to heal.

IMPETIGO

Looking through my records of impetigo in infancy, it seemed justifiable to separate them into 3 groups. The *first* group includes the extensive cases in the first few weeks of life, viz. pemphigus neonatorum and Ritter's disease. In half a dozen of these cases personally treated, none of the dreaded septicaemic complications in Ritter's disease seemed imminent, and I have not seen grounds for the gloomy picture painted in the text-books. While in Ritter's disease the trouble usually starts somewhere on the head or the head mucosae, one of my cases began with a bullous impetigo of the buttocks containing a heavy pure infection of *B. proteus*. In treatment I have found aureomycin surgical powder useful, mainly because it is an antibiotic in powder form and also for the convenient shaker-container in which it is packed. It is doubtless worth while to have cultures and sensitivities of the organisms determined in advance, in case of complications.

The *second* group is the only one where impetigo was present in its typical form, as one sees it in childhood. It started at the end of the infancy period, from roughly the second year onward. The cases here are of the usual sort, running a short and manageable course.

The *third* group of my cases in which the diagnosis of

impetigo was made, was one which lay in the intermediate age-period, between 3 months and 2 years. From the clinical features and course many may regard the patients as having some phase of infected eczema. In some cases an eruptive follicular eczematide and something resembling epidemic follicular keratosis were associated, as well as the commoner infective flexural lesions resembling seborrhoeic dermatitis, For treatment I have been using a watery shake lotion, the formula of which was recommended by Prof. Miescher, containing 0.25% terramycin, 0.5% proflavine and 5% vioform. It forms a yellow cake which absorbs exudate, disinfects, and prevents spread of the infection. If practicable, I combine it with occlusion of the lesions.

SCABIES

The cases I see have sometimes been treated already with penicillin, cortisone and antihistamines without benefit. Most of the infants have acquired the infection from older school-going children in the house who attend to them. Native nurses are in my experience a less frequent source of infection. When there are cases of infected scabies in the house, it may be hard to prove that the baby's trouble is anything more than an infective eczema, which may persist after anti-scabies treatment. The most useful single clinical clue to scabies in infancy is to my mind a septic condition of hands and feet, although the sepsis in itself may hamper recovery of the acarus, which is needed to prove the diagnosis. The most misleading single condition in diagnosis is a papulo-follicular eczematide discussed below.

FOLLICULAR AND LICHENOID SYNDROMES

It has been mentioned above that some of the eczematides may turn into non-erythematous generalized scaling states which may resemble ichthyosis. Here I wish to mention two more predominantly non-erythematous conditions which are not rare, and may offer even more diagnostic trouble.

The simplest is an acute asymmetrical, centrifugal, fawn-coloured follicular-lichenoid and confluent dermatitis in infants of about a year or more. It is probably a poral eczematide. It starts usually on one buttock with a sheet of fine red-brown follicular lichenoid micropapules. Some central clearing with a suggestion of circinate lesions may become evident. Often the trouble then spreads in a diffuse broad zone for a variable distance down one thigh and leg, later involving the opposite buttock and thigh. I have also seen it start on one ankle and spread upward. There is seldom much irritation, at any rate not at first. When one thigh only was involved, I used to think of contact dermatitis to hand cream of the mother from carrying the baby with one arm, but have since abandoned the idea. Loewenthal, in discussing the problem with me, has suggested an infection whose spread was favoured by lying on the affected side in a soiled cot. Questioning the parents with this hypothesis in mind, it appeared that the rash spread down the leg that was usually uppermost in bed. This would relate the rash to sweating in some way. It reminds one of the statement of Kuno, that the body sweats particularly from the side lying uppermost when perspiration is stimulated during recumbency. A lotion of 3% resorcin has readily cured most of my cases, with or without an antibiotic ointment applied overnight.

Another condition of which I have seen a few examples is a fawn-coloured grouped follicular eczematide starting on the upper trunk in children between 1 and 2 years of age. Lichen scrofulosorum or pityriasis rosea may be suggested. Redness is variable. Scattered papules may also be numerous, resembling prickly heat, and changes to a warm climate may indeed induce an attack. In this, as in the foregoing syndrome, some of the larger scattered follicular papules may be more raised and white, and I have several times been deceived into thinking of early molluscum contagiosum or lichen urticatus. The eruption was also sometimes asymmetrical, and could spread over successive regions of the trunk and limbs in a slow progressive march, with progressive clearing in the parts earlier affected. Here again there was a tendency to form a diffuse *craquelé* dermatitis. My impression is that this type of eczematide is slower to spread and slower to heal than the foregoing, more liable to relapse if healed, and more liable to itch severely. These patients gave me no grounds as a rule to think that the disease was one of the ide eruptions.

The cases of both types mentioned here were seen outside the times at which lichen urticatus occurred. Some of these follicular disturbances were remarkably sudden in onset, and cleared within a few weeks. In 2 cases there was slight purpura with fawn-coloured staining of the skin. Epidemic follicular keratosis and purpuric eczematide were at times regarded with favour in the diagnosis of these cases. A focus of impetigo, flexural eczema or a sore throat preceded some of the widespread follicular rashes without one being able to tell what the relation might be. I have seen one or two examples of a similar sudden non-erythematous patchy and diffuse follicular ichthyosis and purpura in older children, which cleared within a few weeks without a definite diagnosis having been made. My patients in this group were treated similarly to those of the foregoing types, but with less success, and in one case meticorten was needed to control the itch.

OTHER CONDITIONS

Ichthyosis. I have seen only a few infants with undoubted ichthyosis vulgaris. These have started during the first year with a picture of follicular ichthyosis. They have not been 'allergic' children. The more typical cases start a little later in life. More common, though still infrequent, has been a picture of generalized *craquelé* dermatitis which has been part of an eczematide of infancy. These eczematides have tended to be of a type resembling pityriasis alba or of the gyrate variety, which have changed either spontaneously or with treatment into a non-erythematous general scaling. The condition is reversible, and is unrelated to true ichthyosis. It is important in the differential diagnosis of the xerodermas of infancy, particularly in the second year of life.

Pityriasis Alba. This complaint has always appeared to me to be an eczematide rather than an impetigo. Ringworm and leprosy have been considered in the differential diagnosis by general practitioners as a rule. In infancy there had not been any foregoing skin dryness. The clinical associations have all suggested a relationship with infantile seborrhoeic dermatitis. The disease may begin with the characteristic scurfy spots on the face and arms within the first year, and I have noted the characteristic deficiency of tanning in the spots at this early period as well. Most mothers have tried emollients and antibiotics without success before I see the baby. The best single treatment is to my mind one of the hydrocortisone-antibiotic combinations locally applied, though sometimes I

have been satisfied in mild cases with a resorcin or a coal-tar lotion. Pityriasis alba is far commoner in older children, and may be seen at times even up to the late twenties.

Sandworm (Ankylostoma Braziliense Larva). As soon as a child in the Transvaal can play outside, he is liable to get sandworm of the buttocks and perineum from sitting in damp sand. Sandpits, building sand, river-shore sand and sifted ground have all been responsible. My youngest patient was 13 months. Since sandworm causes an eosinophilic granuloma going down to the fat, I have used systemic meticorten in short courses with conspicuous lessening of the infiltration and itching in severe infestations. The worm can be killed with Lortat—Jacob's cryocautery provided the advancing end of the track is clear. It is undoubtedly the handiest method of freezing, since it is not very painful, and can be accurately applied to small areas on the perineum with ease. It permits freezing under pressure, and it is hard to believe that a worm larva in the epidermis can withstand —80° C for 10-15 seconds under 1 kg. pressure. Judging by results, 50-80% of tracks are abolished on one such treatment. A total of 1-5 treatments extending over several weeks may be needed to cure a case where the worms are intermittently active and one has to repeat a treatment that was unsuccessful earlier. I can imagine nobody who uses a cryocautery wanting to freeze with ethyl chloride again. Though I have no personal experience of them, the freons as refrigerants seem to share the disadvantages of ethyl chloride.

Geographical Tongue. I have seen 5 infants, 4 of them girls, with typical chronic migrating geographical tongue, and have been thus persuaded that its onset at any time within the first 2 years of life is not unusual. This fact is not mentioned in dermatological works, and the conditions deserves more study at all ages. Histological specimens from adults have shown, in my material, a polymorph-infiltrated necrobiosis in the tongue epithelium at the site of the milky advancing edge of the lesion. One of my infant cases had associated choking attacks from an intermittent oedema of the tongue. No other associated features were noted. I tried meticorten for 4 days (2.5 mg. *b.d.*) in one baby without result.

Nodular Allergide. I have had one case in a girl starting at the age of 6 months and persisting nearly a year, with a clinical picture of fixed white petaloid and papular infiltrations on the legs that became red at times. Clinically the diagnosis of urticaria pigmentosa was suggested, but the biopsy showed a nodular allergide. She had had recurrent tonsillitis since her 11th week.

Ringworm. Cats are the usual source of ringworm in infants, and so far I have isolated nothing but microsporon canis from the few cases seen. The age of onset obviously depends on when the baby first starts playing with the cat.

Alopecia Areata. I have seen a few cases of abortive alopecia areata in children just over 2 years of age. The loss of hair was characteristically incomplete and the patches ill-defined. Many may question the diagnosis.

Angiomas. Relatively few cases of angioma come my way, and I am in some doubt as to how other practitioners in South Africa treat them. A satisfactory situation would be the treatment of these cases by a critical dermatologist versed in radiotherapy who is prepared to follow the cases, publish and openly discuss his results, and work in a centrally placed paediatric clinic. With anything less than this we cannot be sure that the patients will get a square deal. Alternative schemes seem unlikely to succeed fully and an ideal scheme may also not easily be realized. Ever since radiologists found out that they were the country cousins of the atom scientist, therapeutic services have been liable all over the world to vicissitudes and explosions.

Other Tumours etc. The rarer conditions such as urticaria pigmentosa, naevoxanthoendothelioma etc. have no special place here, and everyone has seen his own group of wonders. Other varieties of moles, warts, pyogenic granulomas etc., possess to my mind no extra interest when they occur in infancy.

Urticaria and Angioneurotic Oedema. Urticaria and angioneurotic oedema are disorders seldom referred to me. The infants I have seen have appeared to develop the condition from inoculations and various soothing and teething medicines.

Erythema Multiforme. I have seen 2 severe cases in children of about 2 years of age, with a chronic course and no discoverable cause. The occasional case of toxicoderma from medicaments was also seen.

Pityriasis Rosea, Psoriasis. These conditions are rare, besides being difficult to identify under the age of 2. Several cases of pityriasis rosea may lurk in my group of generalized eczematides, as noted above. I have seen a small number of apparently genuine pityriasis rosea cases in infancy, in one of whom the mother developed the same condition 3 months later.

Acne of Infancy. I have had one typical case in a female starting at 10 months, and distinguished by a particularly slow evolution of the papulopustules over a month or two, before draining and forming depressed scars. There were no discoverable signs of tuberculosis, virilization or contact with oils. The name acne neonatorum is misleading. This disorder has escaped the notice of a recent author on paediatric dermatology despite its having been frequently described. Achromycin syrup worked fairly well in my patient in aborting the deep pustules.

SUMMARY

A survey is presented of the practical problems in the diagnosis and treatment of the dermatoses of infancy as seen in dermatological practice in the Transvaal.

The Central Council for Health Education, London, will hold a Summer School for professional workers in the fields of Health and Education at Bishop Otter College, Chichester, Sussex, England, on 19-29 August 1958. The subject of the seminar will be 'The Science and Art of Health Education'. The course is intended for doctors, nurses, health inspectors, teachers, industrial welfare officers and others concerned with health education. Besides sessional classes a programme of social and recreational activities will be arranged, including visits to places of interest in the district, and also an exhibition of health education material from various countries. Participants will be housed at Bishop Otter College and the inclusive fee for tuition and residence will be £22 0s. 0d., of which £2 2s. 0d. is payable on registration and is not returnable after 19 July except in the case of personal illness attested by medical certificate. Application for enrolment should be made to the Medical Director, The Central Council for Health Education, Tavistock House, Tavistock Square, London, W.C. 1.

DRIED SKIMMED MILKS AND KWASHIORKOR

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In a previous investigation[1] concerning the dietary treatment of kwashiorkor, it appeared that an imported spray-dried acidified skimmed milk was superior to a local roller-dried skimmed milk in initiating cure. The imported milk, however, was supplied in air-proof and moisture-proof containers, whereas the roller-dried milk was packed in bags not impervious to moisture. The possibility that the Maillard reaction[2] had occurred in the latter case could therefore not be excluded. In this reaction, which occurs in the presence of excessive moisture, lysine and possibly other amino acids become inactivated. It was further pointed out that lactic acid was added during the manufacture of the spray-dried milk, whereas in that of roller-dried milk lactic acid was added at the time of preparation of the feeds.[1]

Another factor which may possibly have influenced the results obtained is that the majority of the patients who received the spray-dried milk were treated during the winter months, when infective diarrhoea is not a big problem. We have previously stressed the difficulty in establishing criteria of severity in kwashiorkor[1] and the possibility cannot be excluded that the patients who received the spray-dried milk may have been less severely affected than the patients in the other groups.

Since factors other than the method of manufacture of the dried skimmed milk might have been responsible for the good results obtained with the spray-dried milk it seemed desirable that the earlier results should be confirmed or clarified. The roller process is cheaper than spray-drying, especially in installation cost. At least 15,000 gallons of milk must be processed daily to make spray-drying economical. The erection of large spray-drying plants is thus limited by the supplies of milk available and by transport costs, a major economic consideration. Although roller-drying requires skilled attention for the operation of the driers, this process can be employed economically on a smaller scale (5,000 gallons per day).

As the incidence of protein malnutrition parallels that of poverty,[3] it seemed important to ascertain whether good-quality roller-dried skimmed milk is as effective in initiating cure of kwashiorkor as the more expensive spray-dried variety. The high temperature (about 250°F) prevailing on the drums in the roller process causes considerable denaturation of the protein although the product is exposed to this temperature for only about one second.[4] During this process lysine, and possibly to a lesser extent other amino acids as well, may be damaged.[4,5] During spray-drying heat treatment of the milk takes place at about 130°F and lasts only a fraction of a second.[4] Nevertheless, with the improvements which have been introduced from time to time, drum drying can yield a dried milk of high quality.[4]

MATERIAL AND METHODS

The criteria used in the selection of patients were identical with those described previously.[1,6] Sixty patients suffering from kwashiorkor were divided at random into 3 equal groups, one of which received roller-dried skimmed milk, another spray-dried skimmed milk, and the third spray-dried acidified skimmed milk. No vitamin or other supplement was given.

The 3 varieties of dried skimmed milk were high-quality products. They were manufactured by the usual processes,[4] but according to special directions. The production of the roller-dried milk was performed by one of the authors (S.G.W.). The dried milks were packed in tins, each of which contained approximately 26 lb., and stored at room temperature. Unfortunately, owing to a misunderstanding, the degree of acidity of the spray-dried acidified milk was only about 2% instead of the desired 4%. Lactic acid was therefore added at the rate of 1½ minims of 85% lactic acid per fl. oz. of the reconstituted formula, during the preparation of the feeds for the patients in this group.

Therapeutic Regimen

In the first 12-18 hours after admission, all patients received Hartman's solution with 5% dextrose by mouth, as well as 1-2 g. of potassium chloride, before the milk formulae were introduced. The fluid intake prescribed was approximately 2½ oz. per lb. body-weight per 24 hrs. The 3 dried milks were prepared so as to provide approximately 10 calories per fl. oz. but within a couple of days the feeds were strengthened to 15 calories per fl. oz. (1½ oz. of dried milk per 10 oz. of water). On admission, 1,200,000 units of benzathine penicillin G (Bicillin) were given intramuscularly, and sulphadiazine was given orally for 7 days at a dosage of 1½ gr. per lb. body-weight per day. Broad-spectrum antibiotics were prescribed in a few cases only; intravenous therapy with electrolyte solutions and/or plasma and blood was instituted when indicated.

Criteria of Successful Treatment

The term 'initiation of cure' is used here as previously defined by Brock *et al.*[6] Briefly, it can be said to have taken place when the patient's downhill course has been changed to an upward one. By this time, usually after from 12 to 21 days, rapid increases have taken place in the concentration of albumin in the serum and in the serum amylase activity and the patient has lost his oedema, has become interested in his surroundings, and has regained his appetite.

Analytical Methods

The methods used for the determination of serum proteins and serum amylase activity were identical with those prescribed previously.[7] Nitrogen in the food, faeces and urine was estimated in duplicate by a modification of the macro-Kjeldahl method.[8] The following methods were used for the analysis of the dried skimmed milks:

Moisture: A sample of 3 g. of dried milk was dried at 85-88°C for 6 hours or until constant in weight.[9]

Fat: The method described by Stodt[10] was used.

Total Solubility: The method used was that described in Richmond's Dairy Chemistry.[11]

Degree of Acidity: 4 g. of dried skimmed milk were dissolved in 32 ml. of water and the mixture titrated with N/9 NaOH (1 ml. of N/9 NaOH=0·1% lactic acid).

TABLE I. ANALYSIS OF THE DRIED SKIMMED MILKS

Type of skimmed milk powder	Pasteurisation of fresh milk	Organoleptic examination		Total Solubility %	Analysis by Factory										
		Flavour and taste	Burnt particles and foreign matter		Moisture	Fat %	Acidity		Protein % in solids	Lysine %	Vitamins mg. %			Acidity of	
							Lactic acid	pH in reconstituted milk			Thiamine	Riboflavin	Niacin	powder % lactic acid	fresh milk % lactic acid
Acidified spray-dried skimmed milk	10 min. at 90°C	good	absent	100	3·6	2·0	1·95	6·5	33·8	4·1	·37	·66	·74	2·02	0·14
Spray-dried skimmed milk ..	10 min. at 90°C	good	absent	99	2·5	3·0	1·75	6·6	34·7	4·3	·46	·89	·79	1·66	0·14
Roller-dried skimmed milk ..	No pasteurisation	good	absent	85	5·4	3·4	2·10	6·45	36·4	4·2	·45	·73	1·31	1·68	0·18

pH-Value: This was determined by a potentiometric method.

Protein: A modification of the macro-Kjeldahl method[12] was used.

B-Vitamins: Determinations were made by modified methods as used by Hoffmann La Roche laboratories[13] and the Association of Vitamin Chemists.[14]

Balance Studies

Balance studies, based on the technique of Hansen,[7] were performed on 2 male patients in each group. The studies were commenced on the day after admission and separate collections of urine and faeces were made over a 3-day period. Carmine was used to mark the faeces.

RESULTS

The therapeutic effect of the 3 varieties of skimmed milk were assessed on the basis of their abilities to initiate cure and to increase the serum-albumin concentration and the serum amylase activity and in addition, in those cases where balance studies were carried out, on the basis of their effects on absorption and retention of nitrogen.

Analysis of the Skimmed Milks

The results of analysis of the 3 types of dried milk are shown in Table I. The well known difference in solubility between milks prepared by the two processes is clearly illustrated. The fat content was fairly high in all 2 samples analysed. The riboflavin contents of all 3 types of dried milk were remarkably low. There was no appreciable difference in lysine content between the 3 varieties of dried skimmed milk. This amino acid is liable to be damaged if overheating occurs during drum drying.[3]

Effects of Experimental Diets on Initiation of Cure

The results have been summarized in Table II according to a modification of the method described by Brock *et al.*[6] In this Table *group I* includes all cases where dietary treatment

TABLE II. SUMMARY OF CLINICAL RESULTS

Diet	*No. of cases*	*Group I*	*Group II*	*Group III (Deaths)*
Roller-dried skimmed milk ..	20	14	1	5
Spray-dried skimmed milk ..	20	17	1	2
Spray-dried acidified skimmed milk	20	14	3	3
Total ..	60	45	5	10

was successful and in which cure was fully initiated within 21 days; *group II* all cases in which additional supportive transfusion of plasma and blood was necessary to initiate cure; and *group III* all the cases which died. (No deaths occurred within 48 hours.)

For the purpose of statistical analysis all cases not classed as a group-I cure were regarded as failures. A test of significance between the 2 percentages (14 and 17) $P_1 = 70\%$ $(14 \div 20 \times 100)$ and $P_2 = 85\%$ $(17 \div 20 \times 100)$, showed no significant difference. According to this analysis, therefore, no significant differences could be established between the 3 varieties of skimmed milk in initiating cure.

Effects of Diets on Serum Albumin Concentration and Serum Amylase Activity

In Tables III and IV are shown the average values, and the equations representing the regression lines, for rise of serum-albumin concentration and increase in serum amylase activity during treatment. Application of the t-test revealed no significant differences in the serum amylase activity between

TABLE III. INCREASE IN SERUM ALBUMIN CONCENTRATION DURING TREATMENT

Type of Skimmed Milk	*Average values* (g./100 ml)				*Regression Lines**
	On admission	*1st week*	*2nd week*	*3rd week*	
Roller-dried	1·7	2·3	2·9	3·5	$Y = 1{\cdot}72 + 0{\cdot}60 \times$
Spray-dried	1·85	2·9	3·5	3·5	$Y = 1{\cdot}80 + 1{\cdot}39 \times -0{\cdot}28 \times^2$
Spray-dried acidified	1·7	2·4	2·9	3·4	$Y = 1{\cdot}75 + 0{\cdot}57 \times$

* These equations represent regression lines of rise of serum albumin (Y) against time (X) during treatment with the three varieties of dried milk.

the 3 groups during the 3 weeks of treatment. On admission, the average values for serum albumin concentration of the 3 therapeutic groups did not differ significantly, but one or two weeks after admission the average value for the patients who received the spray-dried skimmed milk was significantly higher than the corresponding values for the other 2 groups. At the end of the third week, however, there were no significant differences between the values obtained for the 3 groups and it seems reasonable to conclude that the rise in serum albumin concentration was satisfactory in all 3 groups. The average values obtained for the patients who received roller-dried milk was almost identical with that for the group receiving spray-dried acidified milk.

TABLE IV. INCREASE IN SERUM AMYLASE ACTIVITY DURING TREATMENT

Type of Skimmed Milk	Average values (Somogyi units)				Regression Curves*
	On admission	1st week	2nd week	3rd week	
Roller-dried	62	117	128	147	$Y=62\cdot8+53\cdot79\times-9\cdot02\times^2$
Spray-dried	52	117	118	119	—
Spray-dried acidified	45	92	105	127	$Y=47\cdot27+44\cdot92\times-6\cdot36\times^2$

* These equations represent regression curves of rise in serum amylase activity (Y) against time (X) during treatment with the different varieties of skimmed milk. Due to a flattening of the curve at the 1st week, a parabola could not be fitted on the values obtained in the spray-dried group.

The Effect on Nitrogen Absorption and Retention

It can be seen from the data in Table V that the amounts of nitrogen retained were high in all 6 patients studied, in spite of the fact that an impairment in the absorption of nitrogen is usually found in acute kwashiorkor,[15-17] As, however, the number of patients studied was small, all that can be said is that nitrogen retention was satisfactorily high, irrespective of the variety of dried milk used.

TABLE V. NITROGEN BALANCE RESULTS

Case	Diet	Weight (kg.)	Nitrogen Intake mg./kg./day	Urinary excretion of Nitrogen mg./kg./day	Faecal excretion of Nitrogen mg./kg./day	Total Nitrogen excretion mg./kg./day	Nitrogen retention mg./kg./day	% Nitrogen retention	% Nitrogen absorption
1	Roller-dried skimmed milk	5·5	695	285	161	446	249	36	77
2	Roller-dried skimmed milk	7·8	726	181	87	268	458	63	88
3	Spray-dried skimmed milk	8·6	797	207	108	315	482	61	87
4	Spray-dried skimmed milk	8·0	523	48	81	129	394	75	85
5	Spray-dried acidified skimmed milk ..	8·0	490	92	132	244	266	54	73
6	Spray-dried acidified skimmed milk ..	10·3	658	187	114	301	357	54	83

SUMMARY AND CONCLUSIONS

Sixty Bantu infants admitted to hospital with kwashiorkor were divided in a random manner into 3 equal groups. During a period of 3 weeks one group was given a roller-dried skimmed milk, another group a spray-dried skimmed milk and the third group a spray-dried acidified skimmed milk.

No significant difference could be detected between the three therapeutic groups as regards initiation of cure or increase in serum amylase activity.

Initially there was a more rapid rise in the serum albumin content among the patients who received the spray-dried milk than among the remaining patients, but after 3 weeks no significant difference could be detected between the values obtained for the 3 groups. Nitrogen balance studies, performed on 2 male patients from each group, showed high retention of nitrogen in all cases.

The results therefore indicate that roller-dried skimmed milk of high quality which has been properly packed and stored can be as effective in initiating cure as spray-dried skimmed milk either with or without added lactic acid. This finding is of economic importance because drum-drying is cheaper than spray-drying.

This paper is published with the permission of the South African Council for Scientific and Industrial Research. The authors wish to express their thanks to Prof. J. G. A. Davel for clinical facilities and advice, to Dr. F. Schweigart and Mr. D. J. de Lange for laboratory help, to Dr. H. S. Steyn for the statistical analysis of results, to the Superintendent of the Pretoria General Hospital for his permission to publish this report, and to Dr. W. I. M. Holman for his interest and advice in connection with the preparation of the manuscript.

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DERMATOLOGISTS AND RADIOTHERAPY*

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1. Dr. Loewenthal's paper is but a continuation of the discussion in the *Journal* during 1950-51 on a portion of Dr. Charlton's paper[2] which dealt not only with skin diseases, but also with radiation therapy in benign conditions generally.

2. Dr. Loewenthal did not give the references to those who took part in the discussion or even to Dr. Charlton's paper but merely referred to 'one radiologist' and 'another radiologist' and 'a dermatologist,' so that radiologists and dermatologists who were present at the meeting in Durban, and many of your readers who wished to do so, could not check Dr. Loewenthal's version of what took place.

3. Why Dr. Loewenthal had to brood for 8 years on this subject before joining the discussion it is difficult to understand. When he had the opportunity 7 years ago to discuss the matter, his only contribution was a few lines of verse, which contained neither medicine nor dermatology nor radiology nor poetry.

The arguments and the terminology used by Dr. Loewenthal are so strikingly similar to the letters by Sulzburger[3] and others in their controversy with Professor Chamberlain, which Dr. Loewenthal calls the 'first attack on dermatologists,' that one cannot help feeling that it was the publication of these letters

*Submitted as a comment on Dr. Loewenthal's Congress paper[1] which was published under the above title in the *Journal* of 28 December, 1957. A summary of longer original article.

which supplied Dr. Loewenthal at last with the ammunition and material for his paper in 1957.

Dr. Loewenthal ignores, for instance, the fact that Dr. Charlton in the techniques he described treated many skin conditions at 60 KV and there was very good reason for his techniques when he used higher KV; but Dr. Loewenthal says that the apparatus of radiologists is unsuitable for dermatological conditions. The so-called superficial skin units which most dermatologists possess go up to 120 KV; only a few have the Dermopan, which goes up to 60 KV. So a dermatologist who has a unit going up to 120 KV can legitimately, according to Dr. Loewenthal, use that unit at 60 KV but, if by any chance the radiologist has a unit which goes up to 200 KV and can still be used at 60 KV, this unit is unsuitable for dermatological purposes. As a matter of fact, most radiologists have special superficial units of the 45 to 60 KV type, and it was Cipollaro himself who in answer to a question by the late William Harris stated that dermatologists could not have 45 to 60 KV units because they had to have units which could cover larger fields and, therefore, used the higher kilovoltage units.

4. In the present article Dr. Loewenthal does not use poetry, but has changed to fanciful prose with references to bull-fights and matadors with which he chooses to describe 'my own contributions to the discussion some 8 years ago,'[4, 5] but again without giving the reference.

5. If Dr. Loewenthal had given the references, one would have been able to see that after appealing for the 'rules of polite debate' he promptly misquotes Dr. Charlton. Dr. Charlton had stated that the X-rays do not kill the spores of fungi. Dr. Loewenthal in his quotation leaves out the 'not' and then on this misquotation proceeds to build up what he considers a case against the treatment by radiologists of dermatological conditions. Even if Dr. Charlton were wrong, does the fact that one radiologist makes a mistake prove anything?

6. Dr. Loewenthal's attack on Dr. Charlton and his misquotations without giving the references will be deplored not only by radiologists but, I am convinced, by all dermatologists. Dr. Charlton retired some years ago after holding the senior radiological appointment at the Johannesburg General Hospital for 16 years and after 25 years as a senior practising radiologist. It is doubtful whether Dr. Charlton, who is no longer in Johannesburg, will ever see Dr. Loewenthal's paper and, even if he did, it is unlikely that he would be bothered to reply to an attack made on an unnamed radiologist.

7. Dr. Loewenthal refers to two attacks made on dermatologists by radiologists. In what he calls the first attack (here he supplies the reference) he gives his version of the controversy between Professor Chamberlain and the American dermatologists. Professor Chamberlain, a recognized authority throughout the world, gave a press interview in 1956 after the Geneva Conference on Radiation of 1955. He made two points. One was that radiation was used far too frequently in benign dermatological conditions and that the dermatologists were the main culprits (up to 99%). His second point was that when radiotherapy had to be given it should be given by radiologists.

Seven senior dermatologists in the US wrote letters to the *Archives of Dermatology* protesting at Dr. Chamberlain's making his views public and, although they resented Dr. Chamberlain's view that radiotherapy should be given by radiotherapists, they resented still more the fact that Dr. Chamberlain said that X-ray therapy was being used far too frequently for benign conditions and that the dermatologists were the main culprits.

The arguments put forward by the dermatologists are embodied in Dr. Loewenthal's paper. They came out with the usual statement (1) that radiotherapists have not got suitable machines for treating skin conditions, (2) that radiotherapists cannot know anything about skin conditions, and (3) that because dermatologists in the US to obtain the Diploma of the American Board of Dermatologists, have to show that they have had 5 years' experience in radiotherapy, the dermatologists are much better equipped to treat patients than radiologists are. Some of the writers of these letters are the very people who warned the American dermatologists not to undertake X-ray therapy unless they were adequately trained. It follows, therefore, that there must be many dermatologists who have not had the 5 years' radiotherapy training mentioned in some of the letters.

Dr. Loewenthal's version of the controversy leaves out completely the point made by Professor Chamberlain that X-ray therapy is used too frequently by dermatologists, physicians and radiologists in benign skin conditions, and that dermatologists were the worst offenders.

One would think from Dr. Loewenthal's version that Professor Chamberlain, whom he belittles with various sneers and to whose status he does not do justice, was completely crushed, but no impartial observer would accept this version.

8. In his version of the second attack, although he refers to an article published in 1949 and the subsequent discussion by radiologists and dermatologists in 1950 and 1951, he does not give the references. The article was written by Dr. Charlton[3] and Dr. Loewenthal merely refers to him as 'a radiologist,' misquotes him, and then goes on to build up a case against radiologists treating skin conditions. He refers to the discussion on this paper by 'another radiologist,' who happens to be myself. I published two letters[4, 5] on the subject, in which I gave a list of some of the hospitals in London and Great Britain in which the dermatologist was not allowed to treat skin conditions by radiotherapy, and in others where the dermatologist was permitted to treat skin conditions in the hospital but only up to a total dosage of 500-600 r. I mentioned that in Sweden dermatologists were not allowed to treat any cases with X-ray, and that at a hospital in Sydney, Australia, the dermatologist prescribed the doses for benign conditions but the radiologist supervised this dosage and malignant conditions were seen by the dermatologist with the radiologist. I also mentioned that Dr. Cooper, of Brisbane, and Dr. Bray, of Sydney, who is in charge of the radiotherapy department, informed me that the dermatologist is not permitted to treat malignant skin conditions. He is only allowed to prescribe up to 500 r for non-malignant conditions.

Dr. Loewenthal's version of this, 8 years later and without giving the reference, was as follows: 'We were given an impressive list of countries in which (so he had been informed) the dermatologist is either forbidden to use X-ray therapy or is limited to using it in small doses. Personal enquiry in many of these countries has convinced me that the radiologist's leg had been pulled with incredible ferocity.' He also goes on to state that 39 out of 41 participant countries stated that the dermatologist had the right to practise X-ray therapy.

Can there be anything more misleading and inaccurate than Dr. Loewenthal's version? I mentioned a number of hospitals in Great Britain and Stockholm and two in Australia. Dr. Loewenthal calls this an impressive list of countries and states that he had made personal enquiry in these coutries. He is careful not to state that he made enquiries in the countries I mentioned. He tells us several times that he had attended the Congress at Stockholm. Will Dr. Loewenthal tell us whether he made enquiries in Stockholm and whether dermatologists there are allowed to use X-ray therapy either in hospital or private practice? Will Dr. Loewenthal state whether he made enquiries at the hospitals I mentioned in London and Great Britain and whether my statement was true or not? Dr. Loewenthal tells us he attended a conference in Great Britain, and so he must have had ample opportunity to find this out. Will Dr. Loewenthal tell us whether he confirmed my statement with the Sydney and Brisbane Hospitals in Australia? He does not tell us whether Australia was one of the countries in which he had made personal enquiry.

Tactics of this description and this method of debate, incidentally after calling for the 'fair rules of debate,' surely cannot be condemned too strongly. It is an insult to people reading this *Journal* to attempt to put such statements across. From whatever angle one looks at it, it must be condemned. Dr. Loewenthal did not give the references; did he bother to read the original letters on which he based this paper?

Every section of his paper contains statements of similar type and value. He tells us for instance 'that one of the justifications for radiotherapy by dermatologists is that dermatologists have been and are still responsible for many advances in radiotherapy in many countries'. He gives a reference presumably to prove this. This reference, *Strahlentherapie*, 1950, cannot be obtained in any library in this country. The article by R. Schmitz to which he refers does not give the advances made by dermatologists; it is a historical review and contains the names of many physicists and radiologists. This reference to R. Schmitz is, however, given in a booklet called the 'A.B.C. of the Dermopan' prepared by Dr. E. H. Graul for the Siemens Co. and given away to customers.

9. These absurd claims for priority for dermatologists and the amount of work they have done on the subject, are taken from the American dermatological literature. For instance, Osborne[6] states,

'Many of the outstanding contributions through the years in radiology have been made by dermatologists'. Many of the American dermatologists make similar statements. It is rather significant, however, that 22 of the 30 references given in this little book on the Dermopan refer to articles in the radiological literature. I do not think it is an exaggeration to say that for every dermatologist who has made any contribution to radiotherapy, there are hundreds of physicists and radiologists who have done so. One is surprised that Dr. Loewenthal does not claim that every dermatologist should be allowed to use radium or radon either as surface applicators or for interstitial use because the first radium burn sustained by Becquerel himself in 1901 was diagnosed as a radium burn by a dermatologist, Besnier.

10. I must draw attention to Dr. Loewenthal's statement: 'I also wish to make it clear that no responsible dermatologist would suggest radiotherapy should be used by those whose specialist training has not included adequate instruction in the subject. I associate myself with those who would forbid the use of radiation therapy to dermatologists not specifically trained in this method.' If that is so, why does he object to radiologists making this statement? Any dermatologist, whether he has had this adequate training or not, may buy an X-ray machine and treat patients. A dermatologist is not compelled to have any X-ray training and can register as a dermatologist without giving evidence of any such training. How then is the patient to know whether a dermatologist using an X-ray machine has or has not had this training in radiotherapy?

11. He tells us he is on the International Committee of the Education of Dermatologists, which apparently decided last year in Stockholm that there should be 3 months' full-time instruction for dermatologists in radiotherapy. It follows, therefore, that hitherto dermatologists have not been compelled to have such 3 months' instruction; nor are they compelled to have it at present. His description of some of the dermatological clinics on the Continent has nothing to do with the question of whether dermatologists on the specialist's register in South Africa are qualified to use X-ray therapy or not. South African dermatologists, with possibly one or two exceptions, were not trained on the Continent.

12. His section on the treatment of skin diseases by radiologists is also a paraphrase of some of the arguments used in the American literature. He again states that he knows of radiologists who have actually treated 'foot ringworm'. It is difficult to understand why Dr. Loewenthal is so virtuous about foot ringworm unless it is but an attempt to use a stick with which to beat Dr. Charlton. The eminent Cipollaro in his letter in the controversy with Dr. Chamberlain mentions the value of X-ray therapy in foot ringworm and the book by MacKee and Cipollaro[7] recommends X-ray therapy for dermatophytoses of the feet under certain conditions; so do some of the radiological books I have quoted. It is difficult to understand how a man of status in his own section of the profession could use futile arguments such as this. Even if there were a radiologist who had used X-ray therapy erroneously, what does that prove?

He cites as the second criticism 'the unsuitability of the X-ray equipment' generally used by the radiologist for the treatment of skin conditions. Dr. Loewenthal's ignorance of radiologists and their apparatus is simply amazing. He does not seem to realize that even a deep therapy unit may be operated at a low voltage suitable for skin diseases. This argument, too, is of course taken from the letters to which I have already referred. He says radiologists' treatment of skin conditions rely 'on heavily filtered penetrating high-voltage radiation which is delivered to deeper structures where it is . . . potentially dangerous'. It is difficult to describe nonsense of this type. Quite apart from the fact that deep-therapy units can generally be operated at low voltages, radiotherapists have other X-ray therapy machines available. It is the dermatologist who has to rely on a single machine.

13. Dr. Loewenthal becomes quite lyrical on the subject of the beryllium window used in the tube for Grenz-ray therapy. He does not seem to know that for years there have been deep therapy machines with beryllium windows available. His enthusiasm for Grenz-ray is unbounded and he refers to a flood of publications, but only gives references which cannot be obtained in this country. An assessment of the value of Grenz rays may be obtained from an address by Professor Pillsbury[8] and the subsequent discussion. There is nothing that a dermatologist can do with Grenz rays that a radiologist cannot do with X-rays. Dr. Loewenthal repeats the argument of the American dermatologists to which I have already referred that the dermatologist knows exactly the depth of the lesion and that the X-ray therapist therefore cannot treat these lesions. It is the extraordinary ignorance of what radiotherapists treat and how they go about it on the part of Dr. Loewenthal and the other dermatologists that makes it quite frustrating and hopeless to debate with them. A radiotherapist is apparently able to treat a carcinoma of the larynx, a carcinoma of the breast or a carcinoma anywhere else and he is expected by the specialists in those particular branches to do so. A radiotherapist can work out the dosage to be delivered in depth to these tumours, but when it comes to the skin he just cannot do it. He cannot look up a chart and see what the penetration is at 50 KV, say, for a certain area and certain focal skin distance. It is just beyond the radiologist's capacity!

He refers to total body radiation at 50 KV and again there is a reference to a journal which cannot be obtained in this country. He does not seem to know that total body radiation has been practised for years, at various voltages and various intensities, such as the Heublein technique at the Memorial Hospital in New York.[9]

Dr. Loewenthal mentions that dermatologists use isotopes and refers to thorium X.[10] The use of thorium X in this country is forbidden without the special permission of the Atomic Energy Board or the C.S.I.R., and there is no dermatologist who could have used isotopes or is qualified to use isotopes in South Africa or would be permitted to use them.

The main issue, however, which Dr. Loewenthal avoids, is that South Africa is different from any other country in having a specialists' register. For every speciality, rules and regulations are laid down restricting practitioners in one speciality to that speciality. A thoracic surgeon is not expected to remove a gall-bladder no matter how much experience he may have had of general surgery before becoming a thoracic surgeon. Similarly, a neurologist may have had as part of his training years of experience as a neuro-surgeon and yet the neurologist is not permitted to operate as a neuro-surgeon. An ear, nose and throat surgeon cannot remove a thyroid for thyrotoxicosis although he removes a larynx for a carcinoma. No specialist in any other branch of medicine would dream of putting up an X-ray apparatus to treat his patients or would be permitted to do so. X-ray therapy is part and parcel of the treatment of carcinoma of the breast; will a general surgeon ever put up an X-ray unit to treat it?

Clinicians of the Memorial Hospital, New York, all had more radiotherapy training than dermatologists are expected to have and yet the scheme for the prescribing by clinicians of their own therapy was a failure and it has been abandoned.

14. The S.A. Medical and Dental Council either did not expect dermatologists to do X-ray therapy or overlooked the fact that they might do it, and therefore did not lay down any regulation that dermatologists who wish to practise X-ray therapy must show proof that they are competent to do it. It is possible for a dermatologist who has not had training in X-ray therapy and is not competent to do it to buy an X-ray machine and proceed to treat his patients merely after reading the little book on the Dermopan or not even that.

The public knows we have a specialists' register. Does it not follow that a patient going to a dermatologist for treatment is under the impression that when a dermatologist uses an X-ray machine he is invariably competent to do so; the patient is thus being misled. The patient thinks that he is being treated by a specialist in radiotherapy. I cannot visualize the dermatologist saying to his patient, 'I am a specialist in dermatology and not a specialist in X-ray but I will give you treatment all the same'.

The dermatologist sees patients who come directly to him as well as those who are referred to him, not necessarily for X-ray therapy. There are, therefore, several objections associated with the scheme of X-ray therapy by dermatologists. The patient may, and frequently does, according to Professor Chamberlain, a radiologist, and Professor Pillsbury, a dermatologist, receive X-ray therapy when it is unnecessary and when the condition could have been cleared up with ordinary dermatological treatments only. Small doses of X-ray given at weekly intervals up to a maximum total of 400 or 500 r will not do the patient any harm and it will never be possible to say whether the patient would or would not have improved to the same extent without X-ray because, in general, the treatment is uncontrolled.[11] The second objection is that a dermatologist, inexpert and untrained, in radiotherapy, may overdo this treatment and do the patient

harm, as Professor Chamberlain, a radiologist, and Professor Pillsbury, a dermatologist, have demonstrated.

15. What is the position with the radiotherapist? A radiotherapist does not see patients directly. They are always referred by another specialist or general practitioner. The patient is therefore assured that X-ray is only being given after every other remedy had failed and that X-ray therapy is the recognized method of treatment for his particular condition. The patient also has the assurance that the radiotherapist has shown the Medical Council that he has had the minimum amount of training and qualifications to undertake therapy, and the patient remains under the control of his own doctor, who may stop the treatment if he is dissatisfied and to whom the radiologist has to report on the progress of the patient.

Dr. Loewenthal's contention that the radiotherapist knows nothing about skin diseases is surely nonsense. The ear, nose and throat man might argue that the radiotherapist knows nothing about carcinoma of the larynx or the surgeon might argue that the radiotherapist knows nothing about carcinoma of the breast. Dermatological conditions are the easiest and simplest to treat with radiotherapy. I have indicated that the vast majority of dermatological conditions are treated by dermatologists with the simple formula of 75 r once a week up to say 500 r, or 200 Grenz rays per week up to 800 r. It is only in localized conditions that more is given. Radiotherapists, as a rule, use much smaller doses than dermatologists. Lectures on radiotherapy of dermatological conditions are given as part of the diploma for radiotherapy and be it noted that at these lectures dermatologists are permitted to attend.

It is abundantly clear that many noted dermatologists and radiotherapists consider that the use of X-radiation in the treatment of skin diseases is badly overdone and more often than not is unnecessary. One need only refer to the article by Twiston-Davies, dermatologist at the Manchester-Salford Hospital for Skin Diseases, to realize the difference of opinion amongst dermatologists on the value of X-ray therapy even in such conditions as eczema, for which X-ray therapy has been such a popular method of treatment. He states for instance: 'I also have the impression that during the war years 1939-45 I did not see a single soldier who was helped by X-ray and that since the war I have seen only one patient with eczema out of a grand total of about 50,000 new cases seen who responded at all impressively.' He summarizes the whole position as follows: 'Disguise or suppression of the truth is bad for science, blunts our own powers of perception, and must even come under the suspicion of being bad for the patient.' It is difficult to believe that with modern views on the value of X-ray therapy in dermatology any private dermatologist can find sufficient patients to treat in his own practice to justify the employment of a full-time radiographer or even the purchase of an X-ray machine.

16. Dr. Loewenthal's affirmation of the necessity for training in radiotherapy does not alter the fact that a dermatologist can give this treatment in South Africa as well as elsewhere, in spite of the specialists' register, without such training. It is time that the Medical Council took cognizance of this position. I know of a number of dermatologists who have had many years of experience at radiotherapy but I am afraid that many dermatologists have not. There are other dermatologists who have courageously refused to buy X-ray machines.

The solution of this problem of the dermatologist and radiotherapy lies in one of the following two directions:

(*i*) That the Medical Council should lay down a standard of radiotherapy for the dermatologist before putting his name on the specialists' register or, as the position is with all other specialities, that the dermatologist should stick to his own speciality, which is dermatology and not radiotherapy.

(*ii*) The Medical Council created a specialists' register for the protection of the public. Why is not this protection extended to the public, therefore, as far as X-ray therapy by dermatologists is concerned? The Atomic Energy Board has also laid down rules to protect the public from unnecessary radiation. Why does it not take action against dermatologists who have used thorium X without permission when it is so anxious that radiologists should observe the rules and regulations pertaining to radio-active substances?

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MEDICAL EDUCATION IN TORONTO

ROBERT M. JANES, *Emeritus Professor of Surgery, University of Toronto; Sir Arthur Sims, Commonwealth Travelling Professor* *

There are certain interesting parallels in the histories of South Africa and Canada. Important Dutch settlement occurred in South Africa in the latter part of the 16th and the early part of the 17th centuries; at about the same time early French settlement was taking place in Canada. Important British settlement in South Africa took place in the latter part of the 18th and early part of the 19th centuries, in which period much the same thing was happening in Canada. When South Africa was discovering its first diamond the provinces in Canada were undergoing federation to form in 1867 the Dominion of Canada.

I suspect that there was a somewhat similar pattern in the diseases which plagued the early settlers. Scurvy was rife on ships coming to Canada and, during the long winter months, in the settlements. It was relieved and later prevented by 'spruce beer' made by boiling bruised branches of the spruce tree in water. It was at approximately the same time that Captain Cook was discovering how to combat scurvy at sea and lime-juice came to be served daily to the British navy. Smallpox which had been brought to Mexico by Spanish troops spread from there to the North American Indians and took a terrific toll in Canada of Indians and white settlers.

Most of the diseases that were common on overcrowded ships of the time were brought to Canada—yellow fever, typhus, typhoid fever, Asiastic cholera, dysentery and diphtheria. There was a tremendous migration to Canada from Ireland after the potato famine and the mortality on the emigrant ships ran from 6 to 12% of the total number of passengers.

HOSPITALS AND MEDICAL EDUCATION

I had expected to find that medical education had been established in French Canada (Lower Canada) much earlier than in Ontario (Upper Canada), but this was not the case. The first Canadian hospital, the Hôtel Dieu in Quebec, was founded in August 1637 by the Duchesse D'Aiguillon and the Augustine Hopitalières of Dieppe. It was the first hospital established in North America north of Mexico and it held its tercentenary celebration in 1939. A little more than 300 years after this contribution to Canada from Dieppe, Canadian soldiers held a less happy rendezvous in the old city. No effort was made by the French prior to the British conquest of Canada to establish a school of medicine in Quebec. The system of apprenticeship, together with an intermittent migration of French physicians to the colony, apparently sufficed to meet the needs of the time.

The Montreal General Hospital was founded in 1818 and here the first effort was made to organize a medical school when, in 1822, Dr. Stephenson began a series of lectures. Two years later the Montreal Medical Institute, the precursor of the medical faculty of McGill, was established. Almost at the same time, 1824, Drs. Rolph and Dunscombe started a school of medicine and a hospital for clinical teaching in St. Thomas, Ontario. A few years later this school was re-established in Toronto. After that several were started in rapid succession—King's College, Toronto, in 1842; L'Ecole de Médicine et de Chirurgie de Montréal in 1843; in Quebec in 1847 the Incorporated School of

* This appointment is made by the Council of the Royal College of Surgeons of England on the recommendation of an Advisory Board consisting of Members from the other Commonwealth countries. Professor Janes visited the Union of South Africa for 5 weeks in February and March 1958.

Medicine, succeeded in 1852 by the Medical Faculty of Laval; and in Upper Canada in 1850 what later became the Medical Faculty of Trinity. Canada now had 12 medical schools, the youngest being at the Universities of British Columbia and Saskatchewan. The first university degree in medicine given in Canada was granted by McGill in 1833.

Toronto

Since I wish to speak to you particularly of my own school, the University of Toronto, it may be of interest to say a few words of the city itself. The water front on Lake Ontario, where the western part of the city now stands, appears to have been a meeting place for Indian tribes before the coming of the white man. There the Humber River empties into the lake and formed a convenient highway down which the red men from the interior came in their canoes to join others who came by way of the lake itself. On this site the French established a trading post in 1749 and called it Fort Rouille. The seat of government of Upper Canada was situated originally at Newark, now Niagara-on-the-Lake, but this was considered later to be too close to the American border for safety and in 1794 Sir John Graves Simcoe moved it to the north side of Lake Ontario and established there the capital of Upper Canada. It was called York in honour of the second son of George III. In 1812 the legislative buildings were burned and the town pillaged by the Americans. In 1834, having attained a population of about 10,000 the place was incorporated as a city, and the name changed from York to the original Indian name Toronto, meaning a 'place of meeting'.

Since the *Toronto General Hospital* has long played an important role in medical education, its history is relevant. In 1812 England was so occupied with her European wars that the attack upon Canada by the United States received little attention and only a comparatively small force of regular troops was available for defence. The independence of the country was preserved through the valiant action of civilian volunteers and lack of whole-out effort by the USA. At that time the Loyal and Patriotic Society of Upper Canada was formed to help care for the sick and wounded. There must have been a garrison hospital, although I have found no record of it, but there was no civilian hospital. The ravages of the conflict emphasized the need for one. At the end of the war the Loyal and Patriotic Society had medals of gold, silver and bronze struck in England for the heroes of the war. Distribution was to have been according to rank but dispute arose about how this was to be done, probably between the regulars and the volunteers, and finally the medals were defaced over an anvil, melted down and sold as bullion. The money realized plus the balance of the funds of the Loyal and Patriotic Society, amounting to about £4,000, was used to establish a general hospital. It was a red-brick, two-storey structure 107 feet long and 66 feet wide, and had a capacity of about 70 beds. In 1824 we find the following note in the local paper: 'The York Hospital is the most extensive building in the Province and its external appearance is very respectable.' Some 399 acres had been appropriated by Order-in-Council in 1817 for the establishment of the hospital. The original structure may be considered, therefore, to have been a monument to the heroes of the war of 1812.

The new building was used little at first because of the local prejudice against being sick in hospital. Fire destroyed the legislative assembly in 1824 and between then and 1829 the new hospital was used to house the legislature. In June 1832 the steamship *Great Britain* dropped anchor in the harbour of York with immigrants on board from England and Ireland and brought with it an epidemic of Asiatic cholera. The disease had reached Quebec and Montreal a few weeks earlier. Thus did the great pandemic, which had commenced in India in 1826 and had appeared in England in 1831, reach Canada. The new building was taken over to house the cholera victims and was used fully as a hospital for the first time. When thinking of the problems of today it is worth while to contemplate what must have been the difficulties of that period. In the year 1832 52,000 immigrants landed in the St. Lawrence and passed through the recently established quarantine station at Grosse Ile. Of these, 11,000 reached Upper Canada. Screening was inadequate because of the lack of knowledge of disease and the manner in which it spread. The majority of the immigrants arrived penniless. The population of the town of York, through which most of those bound for Upper Canada passed, was then less than 10,000 and there were few doctors; 30 years later there were only 35.

Since its original establishment the Toronto General Hospital has moved twice, in 1855 and in 1913. In 1853, in order to finance the new hospital, most of the original grant of land was sold at auction. Lots fronting on King Street, now near the centre of the business section of the city, fetched from 3s. 6d. to 8s. per foot frontage. The same land today is worth $1,500 to $2,000 per foot. When the second hospital was completed in 1855, the population of Toronto was 45,000 and there were 35 practising physicians. For the first time the staff was divided by the Board of Trustees into physicians and surgeons.

In one respect the troubles of all hospitals seem to be similar. Financial problems plagued this one. The city refused to accept responsibility for indigent patients and, since many of its clientele were penniless immigrants, finances reached such a state that between August 1868 and August 1869 the doors were closed to new admissions. Subsequently assistance was obtained from the city and province and with the help of donations from wealthy citizens the hospital gradually increased in size until in 1882 there were 361 public and 39 private beds. It was, however, inadequate to meet the needs of the rapidly growing community and the demands of medical education.

Before another move was undertaken a committee of important citizens representing the public, the hospital and the University of Toronto was set up to study and advise upon the future. It was a far-seeing committee and its recommendations were responsible for what is now regarded as a sound relationship between the University of Toronto and the teaching hospitals. The hospital was to be moved to a new site adjacent to the campus, where all the facilities of the University would be available for the training of students of the early years in the humanities and the basic sciences, and senior students would have ready access to all the opportunities that a great hospital has to offer in the way of clinical training. By the terms of the agreement the professor and head of a university department automatically became head of that department in the hospital. For example, the Professor and Head of the Department of Medicine is Physician-in-Chief of the General Hospital. A committee called the Joint Hospitals Relations Board was set up to control appointments. Recommendations from it must be approved by the Board of Governors of the University and the board of trustees of a hospital.

The original structure, completed in 1913 at a cost of $3,750,000, stood on a square of 9 acres and had 768 beds, of which 570 were devoted to public ward services, and ample facilities for out-patient care. It was described as one of the finest, if not the finest, on the North American continent. In 1930 a wing to accommodate 350 private patients was added and in 1932 another building primarily to house diagnostic radiology and radiotherapy, bringing the capacity to 1,250 beds. An extensive programme of modernization is now nearing completion which will centralize the service departments and increase the bed capacity to 1,750.

Toronto can claim the honour of having had the first *hospital for children* on the North American continent. The first building, containing 11 rooms and having accommodation for 16 patients, opened its doors to the public in 1878. In 1887, the jubilee year of Queen Victoria's reign, a hospital of 270 beds was completed and opened. The citizens donated $20,000 in honour of the occasion. The present structure with a capacity of 650 beds was opened in 1951 and is a magnificent plant. Research and teaching have been stressed in this institution for the past three-quarters of a century and contributions from the members of its staff, many of whom will be familiar to you, have placed it in the forefront of the children's hospitals of the world. Two full floors of the present building are devoted to research.

The federation of teaching hospitals used by the University of Toronto is completed by St. Michael's, which was opened in 1892 and now has a capacity of 900 beds, and the Western, opened in 1896 with accommodation at present for about 700. While the General is regarded as the central unit in this federation and the heads of the university departments are there, all hospitals bear a similar relationship to the University and no appointment can be made to the staff of any of them except through agreement between the hospital and the university.

While *medical research* has, so far as I can discover, always been regarded as important in the Toronto school, it received a great stimulus from the work of Sir Frederick Banting and Prof. Charles Best. In 1922 the Banting Institute, which stands across the street from the General Hospital, was opened. Like

all such establishments it has long since been unable to meet the demands for space. In 1955 the Best Institute of Physiology was built alongside the Banting Institute and this has somewhat relieved the pressure. A Cancer Institute recently completed provides, in addition to all the requirements for therapy, magnificent facilities for fundamental and clinical research in that field. All of these developments are part of the University and its basic departments and sources of expert knowledge are available to those working in any unit.

Let us return for a moment to a consideration of the *proprietary schools*. The Rolph school of medicine was interrupted by the rebellion of 1837. Rolph was one of William Lyon McKenzie's collaborators and as such felt it was wise to leave the country for a time. He returned in 1843 and resumed his lectures. This school became the Toronto School of Medicine, the Faculty of Medicine of Trinity College, and the Medical Faculty of King's College. The last-mentioned was abolished in 1853; the others were finally amalgamated in 1903 under the Faculty of Medicine of the University of Toronto. Several other schools made a temporary appearance but space allows mention only of the Women's College of Medicine, which was opened in 1883 because women could not gain access to the other schools. It was closed in 1906 when women were admitted to the study of medicine in the University of Toronto on an equal footing with men. Its memory is preserved in the name of the Women's College Hospital, a fine modern unit of 300 beds staffed entirely by women and recently accorded limited privileges of university teaching. On the occasion of the union of Trinity and Toronto the inaugural address was delivered by Sir William Osler. It was entitled 'The Master Word in Medicine' and is, I think, one of his greatest. It was the need for grounding in the basic sciences that spelled the doom of the proprietary schools. The expense involved in the provision of the necessary laboratories was beyond them.

One or two other milestones in the history of the school should be mentioned. In 1919 a sum of money was donated to the University by Sir John Eaton to establish and maintain a full-time chair of medicine. Prof. Duncan Graham was its first occupant and thus became the first full-time professor of medicine in the Commonwealth. The following year a full-time chair of surgery was established and Prof. Clarence L. Starr was appointed to it. I should add that in spite of their name neither of these posts is entirely full-time: a small amount of practice is allowed but the responsibilities involved in heading a large department leave little time. In 1910 provision was made for a course leading to the Diploma of Public Health and in 1924, through a generous gift from the Rockefeller Foundation, a School of Hygiene was created and endowed. Its impact, I believe, has been international.

POSTGRADUATE TRAINING

Before the first world war few facilities existed in Canada for postgraduate training in medicine. The majority of our men had gone to Britain or the United States for advanced work in the specialties. At the end of the war it was realized by those responsible that the opportunities in these countries would be taxed to meet the needs of their own graduates and that it would be necessary for us to provide for postgraduate training in Canada. As one of the oldest and largest of our schools the University of Toronto has taken a lead. The first organized scheme for the training of surgeons was inaugurated at the General Hospital by my predecessor, Prof. W. E. Gallie, 28 years ago. What he began as a relatively small endeavour was extended during my regime to embrace all of the hospitals engaged in undergraduate teaching and, in addition, the Veteran's Hospital of some 1,500 beds. The course is administered by a committee of the associate professors of surgery of which the head of the department is chairman. The Committee has under its control over 1,000 public ward surgical beds and perhaps an additional 500 private beds. Training in anatomy, physiology, pathology, and biochemistry is provided by university departments in night classes and late afternoon lectures. Trainees are admitted to the course not only from Canada but from many other countries. Appointments are available for advanced training in all of the surgical specialties. Because of the large number of resident posts in the participating hospitals it is usually possible to place a candidate in a job suitable to his needs. Gradually organized courses of training have been set up in other fields and opportunities for advanced training in all the medical and surgical specialties are now provided.

Graduate education has been influenced profoundly by the *Royal College of Physicians and Surgeons of Canada*. It is now 27 years since the College was incorporated by Act of Parliament. It was founded, I think, because of some dissatisfaction with the qualifications for specialists then in existence. The Royal College in Britain had never pretended to say whether a man was clinically competent but merely that he possessed the necessary academic background. This was misunderstood in Canada and many men who had obtained fellowship but had had little or no practical training were grossly incompetent. This applied particularly to surgeons. The standards of the American College of Surgeons at that time were not regarded as being sufficiently high. The founders of the Canadian College cherished a hope that its degree would not only signify that the holder was trained in the basic sciences but clinically competent. On the whole I believe that the hopes of the founders have been fulfilled. There is no doubt that the College has succeeded in raising the standards of medical practice throughout the country. Since a candidate must meet the standards of training before he is allowed to sit the examination, the requirements have slowly been altered to meet the needs of the various special fields. For example, a man may obtain fellowship in surgery as modified for orthopaedic surgery or urology or neuro-surgery; such men must pass examinations in which half the paper is devoted to general surgery and half to the specialty and must face general surgeons and specialists on their orals. Standards are high and as a result so is the failure rate.

In 1939, because some form of national insurance seemed imminent, the Royal College consented, on the invitation of the Canadian Medical Association, to attempt to say, originally through assessment of credentials and later by examinations, who in the country should be considered specialists. In this the College assumed a great burden but there is no doubt that its action and the desire of men to meet the basic requirements laid down has improved the standards of practice, particularly in other than university centres. Generally speaking, the training demanded one year less than for fellowship and the examinations, both written and clinical, were of a more practical nature, e.g. a knowledge of basic sciences necessary to the intelligent practice of the specialty is demanded instead of the more academic knowledge expected of the fellowship candidate. It was hoped to discontinue at some future time the examinations for certification but it now seems doubtful if this will be possible except in particular specialties such as neurology and neuro-surgery, where it is already being done. The period of training demanded for fellowship is so long that men possessed of a sound practical knowledge can scarcely be denied some recognition and the alternative to retaining this qualification would be a lowering of the fellowship standing. This is undesirable.

The scheme for recognition of advanced graduate training in and knowledge of the specialties that has been outlined is, I believe, in certain respects unique. As happens so often in Canada, there has been an attempt to learn from Britain and the United States and profiting from their experience work out something embodying the good points of each. In the beginning the Canadian College conducted primary examinations but these were discontinued and each attempt to re-institute them has failed. The requirements of the College are very similar to those of the American Boards but we believe that we were fortunate that a central examining body was set up before specialty groups arranged their own. Because of this it has been possible to continue to demand a broad basic training even though the candidate intended eventually to confine his activities within a narrower field.

The great argument for a primary examination, of course, is that it weeds out those who lack the mentality required for advanced training, but many of us wonder if it really works that way. Certainly it has not prevented the training of too many specialists in Britain. Admittedly the weakness of our system is its inability to sort out at any early stage those who have chosen unwisely to do advanced training and will never be able to pass the examinations.

Some of this difficulty will be overcome when prospective trainees and those responsible for providing postgraduate training accept, what is undoubtedly true, that men with a poor academic record as undergraduates will probably not be able to pass fellowship examinations, regardless of the length of time they spend on preparation.

THE CHANGING FACE OF MEDICINE*

L. L. ALEXANDER, *President, Border Branch, Medical Association of South Africa*, 1957

My address deals chiefly with my own observations, thoughts, and impressions in regard to the 'changing face of medicine'. I have divided them into 3 periods, viz. (1) the past, up to the last world war, (2) the period between the two world wars, and (3) the present and the future. If I refer more often to the general practitioner, it is because I belong to that group—the labourers of the profession, on call 24 hours a day and 7 days a week.

Dr. L. L. Alexander

The doctor in the period before the Great War was general practitioner, specialist, and consultant, and was there to do anything and everything. He consulted, operated, and visited. When he was called to see a patient, he invariably went, whether during the day or at night, in wind, rain or hail, on foot, horseback, bicycle or cart, and later in luxury by car, and never did he dream of saying 'I can't' or 'I won't come'. He was the family doctor in all senses of the word—friend, confidant and adviser, beloved, respected and esteemed by all. He knew his patients and their families, and participated in all their joys and sorrows. The familiar black bag of that time indicated a confinement on the go somewhere or other. His first consideration was medicine; his profits came after. There were not many directions to which he could turn for assistance in diagnosis; the laboratories, X-rays, etc. began to come into being after the first world war. The drugs at his disposal were limited to a few recognized cures and placebos and he often had to use his own initiative in treating cases. Many cases were lost undiagnosed. Nevertheless, without the aids that are now available, he did a good job of work. Competition was possibly not so keen, and there was no such thing as unethical conduct. This serpent reared its ugly head in the years between the two world wars, and has kept on wagging it ever since, despite ethical laws.

In those days there was very little exploitation of the doctor by the public or *vice versa*—medical aid societies were things never even dreamt of, and benefit societies were few and far between.

Between the two wars things changed quite a lot. More facilities were available to the doctors—laboratories were springing up, and X-rays aiding the doctors in their diagnosis and treatment. The specialist and the consultant began to put in an appearance at the bigger centres, and doctors were specializing in more and more branches. However, the majority of those specializing returned to general practice. In the thirties the doctors were becoming more money-minded. The family doctor still existed, but competition became keener. Medicines and treatment improved and diseases which hitherto were considered incurable were being treated successfully. More doctors were taking a scientific interest in medicine, with the result that research was now going on in all branches. Medicine and surgery now became divided into general medicine, general surgery, and the specialities, and the ancillary services began to come into the picture. The 'quacks' and their counterparts began their decline but, to my regret, they were never completely eradicated and still exist to-day, though in decreasing numbers as the public became more educated in the right and the wrong approach to the treatment of illness and disease.

With the last world war came a marked change in everything, not least in medicine. A large number of medical men joined the services, and for them medicine took on a new outlook. The general practitioner became the regimental medical officer—family doctor to a regiment of men; others worked in field ambulances, casualty stations, field hospitals, and the lucky ones in the hospitals at the base or at home, seeing all kinds of cases, learning new techniques, and improving themselves all round. Here then started the change over to jealousies, envy, exploitation, lack of ethics, even bribery and corruption.

What of those who stayed at home—someone after all had to attend to those who stayed behind. Work was trebled for these doctors, and they found they were working full time. In some cases, it was a godsend to the doctor who stayed behind and was able to pick up the patients left by the others who had gone on service, and so improved his earnings beyond his wildest dreams. This is where practice became a profit-making business first, and medicine as such took a secondary place. True, it is necessary to make a profit out of doctoring—we all must live—but it brings in all the evils possible when money becomes the be-all and end-all of everything.

Many began clamouring to become doctors. There was more money in medicine than in any other profession or business. But there was one snag—to be a general practitioner meant hard work, and plenty of it, and many of the newer men did not fancy this—rather specialize and earn more and do less seemed to be their motto. What was the result? More specialists than ever before, and the number of specialists is ever increasing; so much so that in the bigger centres there now appear to be more specialists than general practitioners. I am not against specialists; on the contrary, I am very much in favour of good specialists. For the good of medicine, and of the patient, it is necessary to have specialists; but too many specialists in anything is never a good thing.

After the war, there started a big change in the general practitioner, and this has become more and more marked. The family doctor, as such, has almost completely gone; in fact, I think in the larger centres he does not exist. The house doctor has now replaced the family doctor. The closer bonds—the patient's confidence and respect—are not as before between the family and the doctor. In many cases he is still considered a friend of the family and his help will be sought in need or stress; but sometimes a doctor cannot even be certain that he is the only man treating the case. If a patient is not getting well as rapidly as he thinks he should or if he is in any way dissatisfied, it is common practice for him to call in someone else, without consulting the first man. There is an Ethical Code in existence, but this is not something that appears in every doctor's make-up and there is more exploitation than formerly, both by the doctors and the public, and also a great deal of 'farming out' despite the efforts of the Medical Association to eradicate it. So keen is the competition in medicine today, that some doctors will stoop to unethical practices to feather their own nests, usually at the expense of their colleagues, but in the long run to the detriment of medicine and of the public. To-day there exists various groups in all branches of medicine. It is my opinion that it is a good thing to have these specialist groups, but for scientific purposes only, and *not* as bodies to dictate the rules, etc. of the Association, which has to deal with every medical man.

Owing to the ever-growing number of specialists, a large number of these men with higher degrees are reverting to general practice.

Advantages of Modern Medicine

During the war years, introduction of the sulpha drugs initiated the revolution of chemotherapy, the effect of which increased in the years that followed. Then came the greatest boon to general practitioners, the penicillin injection, soon to be followed by the oral antibiotics and others. The doctor of today has great advantages over his forbears, and the numerous new germ killers and advanced laboratory techniques and X-ray services are a wonderful aid. We travel in luxury to-day, covering long distances by car or quicker still by plane, which makes it possible to reach other centres to attend lectures, revision courses, etc. The ancillary services have also improved, and today have become a part of medicine.

Medical Aid and Benefit Societies. These are growing like mushrooms, and have given rise to the biggest controversy in the profession—one on which the Association is split into various camps—those who are against them, those who favour them unreservedly, and those who favour them when they prove their *bona fides* and comply with the rules of the Association. I belong to the third group. I feel that these societies play an important

* Valedictory presidential address, East London, 22 February 1958

part in medicine today, in that they are filling the needs of many people who without them would not be able to afford the treatment they need, or who would find themselves crippled in meeting their medical expenses or would have joined the ranks of the free patients at hospitals. The societies also help to cut down the doctor's bad debts. Nevertheless, as in everything, there are the black sheep in the medical aid and benefit societies who try to exploit the doctors and, if given half a chance, would dictate their own policy to the medical profession. Some societies promise their members the moon and in reality give the smallest stars; and others are so afraid of parting with their funds that they delay the payment of the doctors, and quibble over the fees. These are the points that have to be eradicated and I am sure that it can be done without going to the extreme measures as envisaged by the Southern Transvaal Branch, who are prepared to do without any medical aid or benefit societies. Where an approved medical aid society contravenes the rules of the Medical Association, they should be shown where they are at fault, and if no attempt is made to amend their faults they should lose their recognition, just as at present new applicants are not accepted unless they are prepared to act in accordance with our rules. The cooperation between the Medical Association and the ruling bodies of the medical aid societies has reached a very high standard, and only good can come of the friendly spirit at present prevailing. Provided that the renegades amongst their ranks, and those doctors who in turn exploit medical aid societies, can be made to see the real light, both the doctors and the members will benefit considerably.

While on the subject of medical aid and benefit societies, I would stress once again the harm that is being done to the medical profession by those who put everything in monetary gain, to the exclusion of humanity and the welfare of medicine generally.

Many will not agree with what I have said, but my views are the result of many years of mixing with all types of doctors, and others, and listening to what they say. Everything I have said applies to the Border as well as elsewhere, though possibly to a lesser degree. But one thing I am very pleased to record is the wonderful friendly spirit that exists in East London and the Border between the medical men. Faults there are—but we cannot all be perfect.

Finally I would say that the underlying thought in my address is 'Live and let live'. I ask you to apply this to yourselves, your colleagues, and your patients.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES

Members of the Association are advised to ensure that patients who present themselves as members of medical aid societies are members of societies officially approved by the Association, before applying the preferential tariff.

There are societies in operation whose names do not appear in the official list and whose members should be treated as ordinary private patients.

A list of approved societies is published in the *Journal* after each meeting of Federal Council and the next list is due to appear shortly.

NEW SOCIETIES

The following new Medical Aid Societies were approved by Federal Council at its meeting held in Johannesburg on 23-26 April 1958:

1. Cape Portland Medical Aid Society, P.O. Box 1067, Cape Town.
2. Escom Cape Western Undertaking Medical Aid Society, P.O. Box 117, Cape Town.
3. Irvine Chapman Medical Aid Scheme, P.O. Box 316, Vereeniging.
4. Marley Floor Tile Medical Aid Society, P.O. Box 67, Nigel.
5. Steeldale and Union Joinery Medical Aid Society, P.O. Box 1210, Johannesburg.

SOCIETIES REMOVED FROM THE LIST

The name of the following society has been removed from the list of approved medical aid societies and the members are no longer entitled to the preferential tariff:

Reunert and Lenz Ltd. Medical Aid Society (all branches), P.O. Box 92, Johannesburg.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
21 May 1958

MEDIESE HULPVERENIGINGS

Lede van die Vereniging word aangeraai om seker te maak dat pasiënte wat hulle self voordoen as lede van mediese hulpverenigings werklik lede is van verenigings wat offisieel deur die Vereniging goedgekeur is, alvorens hulle die voorkeurtarief op sodanige pasiënte toepas. Daar bestaan verenigings waarvan die name nie op die offisiële lys verskyn nie en waarvan die lede as gewone private pasiënte behandel behoort te word. 'n Lys van goedgekeurde verenigings word na elke vergadering van die Federale Raad in die *Tydskrif* gepubliseer en die volgende lys sal binnekort verskyn.

NUWE VERENIGINGS

Op sy vergadering van 23-26 April te Johannesburg gehou, het die Federale Raad onderstaande nuwe Mediese Hulpverenigings goedgekeur:

1. Cape Portland Medical Aid Society, Posbus 1067, Kaapstad.
2. Escom Cape Western Undertaking Medical Aid Society, Posbus 117, Kaapstad.
3. Irvine Chapman Medical Aid Scheme, Posbus 316, Vereeniging.
4. Marley Floor Tile Medical Aid Society, Posbus 67, Nigel.
5. Steeldale and Union Joinery Medical Aid Society, Posbus 1210, Johannesburg.

VERENIGINGS VAN DIE LYS GESKRAP

Die naam van die volgende vereniging is van die lys van goedgekeurde mediese hulpverenigings geskrap en lede is nie langer op die voorkeurtarief geregtig nie:

Reunert and Lenz Ltd. Medical Aid Society (alle takke), Posbus 92, Johannesburg.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
21 Mei 1958

PASSING EVENTS : IN DIE VERBYGAAN

Dr. Maurice Weinbren of Johannesburg left for England on 22 May to attend the International Cancer Congress in London. Dr. Weinbren expects to return in August.

* * *

Dr. A. W. S. Sichel has been elected a life member of the Ophthalmological Society of the United Kingdom. The life members of this society are now 14 in number—9 in the UK and 5 in other countries, including 2 in South Africa, viz. Dr. J. Luckhoff and Dr. Sichel, both of Cape Town.

* * *

Dr. A. H. Tonkin, Secretary of the Medical Association of South Africa, whose hobby is scouting, has been appointed Deputy Chief Scout for the Union of South Africa and South West Africa. For the last few years Dr. Tonkin has been Divisional Commissioner for the Cape Western Area.

* * *

Research Forum, University of Cape Town. The next two meetings of Research Forum will be held on Wednesday 4 June and Tuesday 17 June in the large lecture theatre on A Floor, Groote Schuur Hospital, Cape Town. They will both be devoted to a report on a large cooperative study on diet, cholesterol, fibrinolysis and blood coagulation. This has been studied in Bantu males and in White males with and without overt evidence of coronary artery disease. In addition to the speakers, Miss A. Moodie and

Drs. B. Kaplan, H. Nossel and V. Schrire cooperated in this work. The speakers on Wednesday 4 June will be Dr. H. Gordon and Dr. R. Lackner; subject, 'Diet, Cholesterol and Fibrinolysis'. On Tuesday 17 June Dr. C. Merskey will be the speaker; subject, 'Diet, Cholesterol and Coagulation'.

* * *

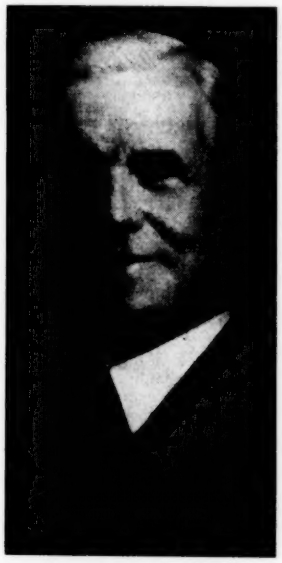
South African Paediatric Association. The next meeting of the Cape Town Paediatric Sub-group will be held on Tuesday 3 June 1958 in the *Lecture Theatre, Red Cross War Memorial Children's Hospital*, Rondebosch, Cape, at 8.15 p.m. Prof. R. Turner, Senior Government Pathologist and Advisor in Pathology to the Union Health Department will speak on 'Immunization'.

Northern Areas Division of the Cape Western Branch. The Annual Dinner has been postponed for one week and will now be held on Tuesday 3 June 1958 at the New National Hotel, Parow, Cape. Tickets may be obtained from the Hon. Secretary, telephone 6-1212.

Die Afdeling Noordelike Gebiede van die Tak Weskaapland. Die Jaarlikse Dinee is vir een week uitgestel en sal nou op Dinsdag 3 Junie 1958 in die New National Hotel, Parow, Kaap, gehou word. Kaartjies kan verkry word van die Eresekretaris, telefoon 6-1212.

PRESENTATION TO PROF. M. R. DRENNAN

On Friday 25 April, a dinner was held at the Vineyard Hotel, Newlands, in honour of Prof. and Mrs. M. R. Drennan. A cheque of £413 was presented to Professor Drennan by Prof. F. du T. van Zyl, Chairman of the Professor Drennan Presentation Fund, which was formed and subscribed to by Professor Drennan's past students and a few colleagues. Professor van Zyl indicated that a large number of the medical personalities and senior anatomists not only of South Africa but also of Great Britain and the United States of America were at one time trained by Professor Drennan. Such names come to mind as Sir Solly Zuckerman, Profs. Sol Cohen, T. Dry, M. Tyler (Nobel Prize Winner), D. Slome, T. Levitt, S. F. Oosthuizen and H. Zwarenstein. This presentation was a token of appreciation of the esteem and high regard held for Professor Drennan. Every medical student fondly remembered the masterly presentation of the anatomy lectures, the vivid and artistic blackboard illustrations, the delightful repetition of phrases, and the inevitable call from the door 'Arrrchie, where are the bones'? Unforgettable also was his approachability by the students at all times. In fact, Matthew Robertson Drennan was an indissoluble part of the history and tradition of the University of Cape Town Medical School. Professor van Zyl also outlined Professor Drennan's distinguished academic career and mentioned his broad interests in anatomy and anthropology and in the preservation of Historical Monuments in South Africa. Last year was the crowning of his academic life, when he was presented with the South Africa Medal by the South African Association for the Advancement of Science and the University of the Witwatersrand honoured him with an Honorary D.Sc. A true South African, he has devoted and still is devoting all his energies towards the improvement of our medical and cultural standards.



Prof. M. R. Drennan

In thanking the past students, Professor Drennan stated that it would be his desire to hand the cheque over to the University of Cape Town to be used in some suitable form for the welfare of the medical students and the Anatomy Department. Professor Drennan recalled the early days of the Medical School and the early bastions of the School—Professors Falconer, Saint and Crichton. He expressed the great satisfaction with which he noted the outstanding careers of his old students. He presented the cheque to Mr. J. P. Duminy, Principal and Vice-Chancellor, who indicated that a Committee would be set up to formulate a method whereby the money would be devoted to its best use and at the same time link Professor Drennan's name with it.

Among the past students and colleagues present were Professor and Mrs. Forman, Prof. and Mrs. H. M. Robertson, Prof. and Mrs. H. Zwarenstein, Prof. and Mrs. J. T. Louw, Prof. and Mrs. J. Kirsten, The Registrar of the University of Cape Town and Mrs. J. P. Benfield, Dr. and Mrs. A. V. Carter, Dr. and Mrs. R. Singer, Dr. and Mrs. E. N. Keen, Dr. and Mrs. P. J. M. Retief, and Drs. T. J. Dry, W. Lennox Gordon, W. Wilkie and A. C. Grobbelaar.

After the presentation, the Principal called a meeting of a small committee—the Dean of the Faculty of Medicine (Dr. B. Bromilow Downing), Prof. M. R. Drennan, Prof. L. H. Wells and Dr. R. Singer and it was decided to establish The M.R. Drennan Fund, which is open for further subscriptions. The interest from the invested capital will be used for (1) providing assistance to needy and promising second-year medical students and (2) providing *ad hoc* grants to distinguished visiting scientists to deliver the 'Drennan Lectures'. If the subscribed capital is large enough, it is also intended to design and construct stained glass windows in the Anatomy Department. The Anatomy Museum will be named the M.R. Drennan Museum. It is hoped that those past students and colleagues whose names have been omitted in the previous appeals will thus have an opportunity of making donations to the M.R. Drennan Fund, University of Cape Town, for permanently linking Professor Drennan's name with the Department which is so indelibly stamped with his character.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

'MAREZINE' INJECTION

Burroughs, Wellcome & Co. (S.A.) Ltd. announce the introduction of 'Marezine' Brand Cyclizine Lactate Injection to sup plement the already established oral anti-emetic 'Marzine' brand Cyclizine Chloride tablets; and supply the following information. Each 1 c.c. ampoule contains 50 mg. of the active principle for intra muscular injection.

The new product is indicated for the treatment of severe, purposeless nausea and vomiting when the oral route cannot be used or is considered unsuitable. It is also indicated when a rapid onset of action is desired. 'Marezine' has proved especially valuable in the prevention of post-operative nausea and vomiting, when 50 mg. (1 c.c.) may be given by the intramuscular route with the pre-operative medication or 20-30 minutes before the expected termination of surgery. 'Marezine' may also be expected to be highly effective in severe cases of vomiting during pregnancy.

The *British Medical Journal* (22 March 1958, p. 675) observes that the most frequent side-effect of morphine is not respiratory depression but nausea and vomiting, which may be 'completely controlled by the use of cyclizine chloride'.

COSCOPIN COUGH LINCTUS AND LOZENGES

Evans Medical Supplies announce Coscopin for effective contro of cough, and supply the following information.

The active ingredient of Coscopin is noscapine, formerly known as narcotine. Noscapine is an opium derivative but is not related chemically or pharmacologically to morphine. It has a powerful specific depressant action on the cough reflex. It is as active as

codeine in the suppression of cough but, unlike codeine, it is a mild broncho-dilator and respiratory stimulant and is non-constipating. It has no analgesic, euphoric or narcotic effect.

Cosopin is indicated for the relief of cough due to a variety of causes, including bronchitis and infections of the upper respiratory tract, post-influenzal irritation and neoplasm. Because of its mild broncho-dilating action and its stimulating effect on the respiratory centre, Coscopin is also of particular value in conditions such as asthmatic bronchitis and whooping cough, where the use of broncho-constricting drugs such as codeine is contra-indicated.

Coscopin is available in two forms—as a palatable cherry-flavoured linctus and as lozenges. Coscopin Linctus is packed in bottles of 4 fl. oz. containing 12·5 mg. of noscapine per dram. Coscopin Lozenges each contain 25 mg. of noscapine and are packed in cartons of 20.

The recommended dosage is: For adults 2 teaspoonfuls of linctus or 1 lozenge every 2-3 hours; for children, 5-14 years half the adult doze, over 14 years as for adults.

Coscopin Linctus should be given undiluted; the Lozenges should be allowed to dissolve slowly in the mouth.

Evans Medical Supplies, P.O. Box 6607, Johannesburg.

DESMOID PILLS 'POHL'

These pills, manufactured by G. Pohl-Boskamp, Hohenleckstedt, Holstein, who supply the following information, are designed for tubeless gastric analyses in accordance with Sahli's method. They consist of a midget capsule (about 1/12 inch in diameter) of rubber, filled with methylene blue and closed with a catgut thread. The capsule only opens when the catgut is dissolved by gastric juice, e.g. if free hydrochloric acid is present in the stomach. If this is the case, the urine will be blue in a maximum of 20 hours, usually after 3-4 hours, as a result of the absorption of the dye. If there is no free acid in the stomach, the pill passes into the duodenum without dissolving and no blue colouring occurs.

Packings: 4 capsules or 15 capsules.

Distributors: Luisen Apotheke (Pty.) Ltd., Windhoek, SWA.

Frisch, H. (1956): Ärztl. Lab., No. 9/10.
Heinkel and Baumer: Ärztl. Prax., 153, 17.
Lipp, J. (1936): Münch. med. Wschr., No. 5.
Lutz (1957): Fortschr. Med., No. 4.
Rühling, O. (1957): Ärztl. Wschr., No. 2.
Sahli (1905): (Uber Prüfung des Magenchemismus. Die Desmoidreaktion, eine neue Untersuchungsmethode.) Korresp. Bl. schweiz. Ärz.
Tengberg (1957): Särtryck ur Svenska Läk.-Tidn., 1957, No. 45.

REVIEWS OF BOOKS : BOEKRESENSIES

HAM'S HISTOLOGY

Histology. Third Edition. By Arthur Worth Ham, M.B., F.R.S.C. Pp. xv+894. 582 Figure Numbers, including 8 Plates in Color. 80s. net. London: Pitman Medical Publishing Co., Ltd. 1957.

Contents: Part I. What Histology Is and How It is Studied. 1. Histology and Its Relationship to Other Subjects. 2. How Histology Is Studied: Ordinary Histologic Methods. 3. How Histology is Studied: The Study and Interpretation of Sections. 4. Electron Microscopy and Other Special Methods. *Part II. Cells, Intercellular Substances and Fluids.* 5. Cells. 6. Intercellular Substances. 7. Tissue Fluid. 8. The Cells of Blood. 9. The Cells of Blood (continued). 10. Platelets and Fibrin. *Part III. The Four Primary Tissues and Their Subdivisions.* 11. The Four Primary Tissues of the Body. 12. Epithelial Tissue. 13. Epithelial Tissue (continued). 14. Connective Tissue. 15. Cartilage. 16. Bone. 17. Joints. 18. Hemopoietic Tissue. 19. Hemopoietic Tissue (continued). 20. Muscular Tissue. 21. Nervous Tissue and the Nervous System. *Part IV. The Histology of the Systems.* 22. The Circulatory System. 23. The Integumentary System (The Skin and Its Appendages). 24. The Digestive System. 25. The Respiratory System. 26. The Urinary System. 27. The Endocrine System. 28. The Female Reproductive System. 29. The Male Reproductive System. 30. The System of Sensory Receptors. Index.

In its third edition this classic work contains much new information derived from electron microscopy. A new section of the book gives a simple account of the principles of this technique and of other specialized methods of histological investigation, including interference and phase-contrast microscopy. Another subject now dealt with in more detail than in previous editions is tissue transplantation.

Clear descriptions of the relation of structural details to function have always been a feature of Ham's Histology and this approach is well sustained in the latest edition. On topics such as muscle contraction and blood coagulation, where new concepts have recently been introduced, the book is commendably up to date. Each chapter is followed by a well-selected list of references to original papers.

Its size, although the third edition is only 30 pages longer than its predecessor, may deter some from reading the book, but those who abstain will miss a most rewarding experience. The student at any stage in his career, or the University teacher, will find in Ham's histology a clear, comprehensive, and up-to-date account of modern histology both as a science in its own right and also in its relation to other medical sciences. It remains the first choice of textbooks for the serious student of histology.

A.W.S.

A SYNOPSIS OF OTORHINOLARYNGOLOGY

A Synopsis of Otorhinolaryngology. By John F. Simpson, F.R.C.S., Ian G. Robin, F.R.C.S. and J. Chalmers Ballantyne, F.R.C.S. With a section on Neurology of the Ear, Nose, and Throat by Charles Harold Edwards, M.R.C.P. Pp. xii+455. 88 Illustrations. 42s. Bristol: John Wright & Sons Ltd. 1957.

Contents: Preface. *Part I. The Ear. Section I.* Surgical Anatomy. *Section II.* Audiology. *Section III.* Equilibrium. *Section IV.* Diseases of the External Ear. *Section V.* Diseases of the Middle Ear Cleft. *Section VI.* Diseases of the Otic Capsule. *Section VII.* Diseases of the Inner Ear. *Part II. The Nose and Paranasal Sinuses. Section VIII.* Surgical Anatomy. *Section IX.* Applied Physiology of Nose and Paranasal Sinuses. *Section X.* Diseases of the Nose and Paranasal Sinuses. *Part III. The Pharynx. Section XI,* Surgical Anatomy. *Section XII.* Applied Physiology of the Pharynx. *Section XIII.* Diseases of the Pharynx. *Part IV. The Oesophagus. Section XIV.* Surgical Anatomy and Applied Physiology. *Section XV.* Diseases of the Oesophagus. *Part V. The Larynx. Section XVI.* Surgical Anatomy. *Section XVII.* Applied Physiology of the Larynx. *Section XVIII.* Diseases of the Larynx. *Part VI. The Trachea and Tracheo-Bronchial Tree. Section XIX.* Surgical Anatomy. *Section XX.* Applied Physiology of the Trachea and Bronchi. *Section XXI.* Diseases of the Trachea and Bronchi. *Part VII. Neurology of the Ear, Nose, and Throat. Section XXII.* Applied Anatomy and Physiology of the Nervous System. *Section XXIII.* Diseases of the Nervous System in Relation to Otorhinolaryngology. Index.

This volume is a welcome addition to the well-known 'Synopsis' series and an outstanding addition to the available literature in the field of Otorhinolaryngology. A speciality in which the majority of text books have in the past been noted mainly for their 'woolly' and untidy nature.

The book is a precise, accurate and rapid means of reference and revision. Diagrams are clear and explicit. It is complete and lacks only the details of operative technique. It is up to the minute and includes reference to stapedolysis, myringoplasty and tympanoplasty and the use by the authors of the term 'Systemic Disinfection' to cover all current antibiotics in vogue should keep it from dating for a considerable time.

It is of especial value to the postgraduate student for higher degrees or diplomas in the speciality. It is of definite reference value to the busy general practitioner, a high proportion of whose work includes complaints referable to the upper respiratory tract.

The book is divided into parts on anatomical basis, the first sections of each being devoted to the relevant anatomy, anatomical principles of surgery, applied physiology and radiology and these are first rate. The last section on diseases of the nervous system in relation to Otorhinolaryngology is by the consultant neurologist to the Royal National Throat, Nose and Ear Group of hospitals in London and is of outstanding merit. Future editions of this book might profitably include a section on stridor in children.

At a very reasonable price it should be read by all interested in this speciality.

G.B.

TUMORS OF THE SOFT SOMATIC TISSUES

Tumors of the Soft Somatic Tissues. By George T. Pack, M.D., F.A.C.S. and Irving M. Ariel, M.D., F.A.C.S. Pp. xvi+820. Illustrations. $30.00. New York: Paul B. Hoeber, Inc. 1958.

Contents: Collaborating Authors. Preface. Acknowledgments. Section I. Classification and Natural History of Tumors of Soft Somatic Tissues. Section II. General Principles of Treatment of Tumors of the Soft Somatic Tissues. Section III. The Treatment of Specific Tumors. Section IV. Sarcomas of the Soft Somatic Tissues in Infants and Children. Section V. Regional Anatomic Considerations in the Treatment of Tumors of the Soft Somatic Tissues. Section VI. Prognosis. *Index.*

This magnificently produced book has a master surgeon as its chief author. The work of Dr. George Pack on malignant tumours is so well known and his reputation so firmly established that one can expect a great deal from a book bearing his name, and in this one is not disappointed.

The pathogenesis, pathology and outlook in the more common and rare tumours are all illuminated in the light of his great experience of over 25 years. The production of the book leaves nothing to be desired. The text is clear and unambiguous and the illustrations really do illustrate.

'The book explains step by step how each type of tumour should be handled, from the initial clinical evaluation of the patient to the performance of the biopsy and the institution of the therapeutic plan, and discusses the anticipated prognosis.'

Divided into 6 sections, Section I deals with the natural history of tumours of the soft somatic tissues. Section II discusses the technique of wide local excisions, resections of the tumour and its draining glands *en bloc* as well as amputations. Section III discusses the treatment of specific tumours. Section IV is concerned with the sarcomas of infancy and childhood. Section V discusses the principles of treatment of those tumours which occur in the neck, the abdominal wall, the buttocks and the retroperitoneum. Section VI summarizes the end results of treatment with an analysis of the factors underlying prognosis.

The question of whether a benign somatic tumour ever becomes malignant is discussed in detail as is whether a sarcoma metastasizes to the local glands.

It is the sort of book that has all the marks of a classic and will have to be kept on the shelves of every clinic dealing with malignant tumours and of every practising surgeon.

S.S.S.

MODERN SEX LIFE

Modern Sex Life. A completely revised and rewritten edition, based on the book *Modern Sex Life*, first published as *Sex Power in Marriage*. By Edwin W. Hirsch, M.D. Pp. 150. New York: The New American Library. 1957.

Contents: Introduction. 1. The Male Sexual Apparatus. 2. The Female Sexual Apparatus. 3. Sexual Frigidity. 4. Prematurity. 5. Sexual Nervousness and Sexual Fear. 6. Unilateral Sexual Attraction in the Male (Male Homosexuality). 7. Unilateral Sexual Attraction in the Female (Female Homosexuality). 8. Men with a Penchant for Feminine Attire. 9. Advice for Young Marrieds. 10. Case Histories. 11. Modern Sex Life. Cardinal Principles of Psychomatics.

This little book may offend some people by its outspokenness. It does, however, fill a want and is very suitable for young people and newly marrieds who come to the doctor for advice for what is at that moment the most important aspect of their lives.

The colloquialisms can be forgiven. Some terms, in spite of the educational effects of American films, will still require to be translated into the King's English, but the basic idea behind the book is a sound one. It explains sex in simple terms and the book can be recommended to general practitioners and gynaecologists.

T.S.

PAEDIATRICS FOR NURSES

Paediatrics for Nurses. 2nd Edition. By Arthur G. Watkins, M.D., F.R.C.P. Pp. 200. 22 Figures. 15s. 0d.+10d. Postage. Bristol: John Wright and Sons Ltd. 1958.

Contents: I. The Child and the Hospital. II. Social Paediatrics. III. Growth and Development of the Normal Child. IV. Mortality, Morbidity, and Prevention. V. Breast Feeding. VI. Artificial Feeding. VII. Weaning, Mixed Feeding, and Diet Tables. VIII. Diseases of the Newborn. IX. Alimentary Disorders in Infancy. X. Deficiency and Nutritional Diseases. XI. Metabolic Disorders. XII. Endocrine Disorders. XIII. Juvenile Rheumatism and Rheumatoid Arthritis. XIV. Tuberculosis. XV. Allergic Disorders. XVI. Disorders of the Mouth and Oesophagus. XVII. Disorders of the Alimentary Tract. XVIII. Disorders of the Liver. XIX. Disorders of the Genito-urinary System. XX. Disorders of the Upper Respiratory Tract. XXI. Disorders of the Lower Respiratory Tract. XXII. Disorders of the Heart and Circulation. XXIII. Disorders of the Blood, Spleen, and Glands. XXIV. Disorders of the Nervous System. XXV. Mental Deficiency. XXVI. Functional Nervous Disorders. XXVII. Disorders of the Skeleton—Muscle, Bones, and Joints. XXVIII. Venereal Disease. Index.

The first edition of this book appeared in 1947 and sought to present to the nurse the main paediatric problems she would meet during her training. It set out to be essentially clinical in its approach and only touched on medical treatment; it omitted nursing techniques and the infectious fevers as it was felt that these were adequately treated elsewhere.

After 10 years the changes and advances in paediatrics made revision necessary. New diseases and syndromes are described in the present edition and others now more fully understood are elaborated. The changes in treatment and prognosis resulting from chemotherapy and the antibiotics are noted and reference is made to the trend towards more prevention by immunization.

The present tendency to carry out more ordinary treatment in the home and for hospital care to be extended to congenital conditions and the rarer metabolic diseases is mentioned and two new chapters have been added to remind nurses that hospitals are not the only agents for the care of the sick children and that when they have to be admitted they are subject to dangers both physical and mental.

The nurse interested in the care of sick children will find this a useful addition to her bookshelves.

H.T.

ESSENTIALS OF CHEMICAL PATHOLOGY

Essentials of Chemical Pathology. By D. N. Baron, M.D. Pp. xii+247. 28 Figures. 25s. net. London: The English Universities Press Ltd. 1957.

Contents: I. Water and Electrolytes. II. Acidosis and Alkalosis. III. Carbohydrates. IV. Proteins. V. Lipids. VI. The Endocrine Glands. VII. Calcium, Phosphorus and the Bones. VIII. The Liver. IX. The Kidneys. X. The Alimentary Tract. XI. The Cerebrospinal Fluid. Appendices. Index.

This book is based on lectures and tutorials given by Dr. Baron at the Middlesex and Royal Free Hospitals to medical students and postgraduates. He felt that there was room for a small book to help the student and junior practitioner to apply his (or her) knowledge of biochemistry to clinical problems and to guide them to the best use of chemical pathology in the investigation and treatment of individual patients. Progress in electrolyte control, for instance, makes a practical knowledge of this essential for the efficient houseman. In this and similar difficulties it should prove very helpful.

It is not a book intended for the chemical pathologist. It does not give details of laboratory methods, but there is a short account of sideroom tests and of the part which the ward staff must play in the conduct of more elaborate investigations. It covers a great deal of ground, much of it very well, but of necessity in so small a book many topics are hardly more than mentioned. Such brevity is good but assumes a good foundation in physiological chemistry and needs often to be supplemented by further reading; references are not given and this is a definite defect. Instead a list of tomes for consultation is given in an appendix with a few lines describing their contents; this may be useful but is a poor substitute for proper references. The index is good and should lead quickly to the information provided.

A few corrections are needed. For example on one and the same page it is stated that the urine phosphorus is low in hyperparathyroidism and that the high urine calcium and phosphorus cause the characteristic polyuria. Such errors are a pity but exceptional.

A good small book at a very moderate price.

G.C.L.

HEADACHE

Headache—Diagnosis and Treatment. 2nd Edition. By Robert E. Ryan, B.S., M.D., M.S. (in Otolaryngology), F.A.C.S. Pp. 421. South African Price: £2 17s. 6d. St. Louis: The C. V. Mosby Company. 1957.

Contents: 1. Introduction. 2. The Physiologic Basis of Head Pain. 3. Objectives of all Headache Treatment. 4. Histamine. 5. Tranquilizing Drugs. 6. Differential Diagnosis of Head Pain. 7. History Taking. 8. Examination of the Headache Patient. 9. Histaminic Cephalalgia. 10. Migraine. 11. Abdominal Migraine. 12. Ophthalmic Migraine. 13. Tension Headache. 14. Generalized Vasodilating Headache. 15. Psychogenic Headache. 16. Nasal Sinusitis. 17. Sluder's Syndrome Headache. 18. Myalgia of the Head. 19. Mixed Type of Headache. 20. Head Pain of Otologic Origin. 21. Temporomandibular Joint Syndrome. 22. Acute Meningitis. 23. Trigeminal Neuralgia. 24. Glossopharyngeal Neuralgia. 25. Facial Neuralgia. 26. Temporal Arteritis. 27. Brain Tumor Headache. 28. Brain Abscess. 29. Subarachnoid Hemorrhage. 30. Posttraumatic Headache. 31. Subdural Hematoma. 32. Lateral Sinus Thrombosis. 33. Alcoholic Headache. 34. Hypoglycemic Headache. 35. Cardiovascular Renal Headache. 36. Constipation Headache. 37. Headache Due to Bone Disease. 38. Headache Due to Blood Abnormalities. 39. Gynecologic Headache. 40. Headache Due to Intoxicants (Poisonings). 41. Headache Due to Cardiac Diseases. 42. Headache Due to Diseases of Endocrine Glands. 43. Headache Due to Cervical Pathology. 44. Headache Due to Cerebral Pathology. 45. Headache Due to Infectious Diseases of Bacterial Origin. 46. Headache Due to Diseases of Virus Origin. 47. Headache Due to Infectious Diseases of Rickettsial Origin. 48. Headache in Diseases of Mycotic Origin. 49. Headache in Diseases of Protozoan Origin. 50. Various other Conditions Associated with Headache. 51. Allergic Headache. 52. Oral Cavity Head Pain. 53. Muscle Tension Headache. 54. Migraine in Children. 55. Postspinal Puncture Headache. 56. Ophthalmologic Head Pain.

This is one of those books that earns neither high praise nor strong criticism. It is a very pleasant and fairly comprehensive work, but it says nothing really new, and nothing that really justifies the publication of yet another monograph on headache. The approach is very personal, with many examples from the author's practice. It is very obvious that the author received much of his training at the Mayo Clinic, which explains why Horton's histaminic cephalgia receives undue emphasis.

The emphasis is very much on treatment, which receives very thorough attention: at times the amount of detail is frankly irritating. The quality of the clinical analyses is usually adequate but somewhat uneven. Even allowing for the sub-title, there is too little discussion of aetiology and pathology, from which a sound appreciation of the other sections could more easily follow.

It is difficult to find a place for this book, not because it is poor, but because its subject matter has been better considered elsewhere; it is not good enough to compete with rival sources of information on headache. In most cases the practitioner will do at least as well to consult a textbook of general medicine. However, the fact that this is the second edition of Dr. Ryan's work suggests that many readers disagree with this reviewer.

K.D.M.

PSYCHOLOGY FOR NURSES

Psychology and Psychological Medicine for Nurses. By Portia Holman, M.A., M.D., M.R.C.P., D.P.M. Pp. 156. 10s. 6d. net. London: William Heinemann—Medical Books—Ltd. 1957.

Contents: Part I. The Basis of Mental Health. Introduction. I. The Basis of Mental Health. II. Mother and Child. III. Relationship with Father and Family. IV. The School Child. V. Puberty and Adolescence. VI. The Young Adult. VII. The Young Adult at Work. VIII. Middle Age. IX. Old Age. *Part 2. Mental Ill Health.* X. Mental Ill Health. XI. Psychosis and Neurosis. XII. Treatment of the Mentally Ill. *Part 3. Human Behaviour in Illness.* XIII. Illness in Childhood. XIV. Illness in Adults. Index.

In this new book the author has attempted to supply the need of nurses for some knowledge of psychology and psychological medicine. She has avoided formal or academic psychology and concentrates attention more on human development and on the behaviour of human beings in the family and society at different stages of their lives.

Normal development is contrasted with deviations from the normal and the second section deals with mental illness and modern methods of treating the mentally ill. Emphasis is placed on the way experiences, either good or bad, influence later development and the last section shows what the nurse must learn to expect in the reactions of human beings to illness and how they must be understood.

The book is written simply and clearly and makes interesting reading.

T.A.

CORRESPONDENCE : BRIEWERUBRIEK

OPKNAPPINGSKURSUS, BLOEMFONTEIN

Aan die Redakteur: Gedurende die week 21-25 April is 'n opknappingskursus vir algemene praktisyns in die Nasionale Hospitaal te Bloemfontein gehou.

Die organiseerders hiervan het gepoog om al die verskillende vertakkinge van die mediese wetenskap te dek deur middel van lesings, saalrondtes, voorgeboorte-ginekologiese klinieke, teaterbesoeke, praktiese en radiologiese demonstrasies, filmvertonings ens. ens.

Sover moontlik was onderwerpe en probleme gekies, waarmee die algemene praktisyn daagliks te kampe het, bv. lae rugpyn, kroniese bronchitis, diarree in kinders, pelviese infeksie, otitis media ens. ens.

Die uitstaande kenmerke van die kursus was die uitstekende organisasie en bekwame dosering aan die kant van die verskeie spesialiteite en outoriteite.

Die algemene mening is dat sulke opknappingskursusse meer dikwels gehou moet word ten einde meer algemene praktisyns die geleentheid te bied om dit by te woon.

Bloemfontein vanweë sy sentrale ligging en baie beskikbare materiaal is die aangewese plek vir opknappingskursusse en selfs vir 'n nagraadse studie-sentrum.

Ondergetekendes wil net baie sterk aanbeveel dat alle algemene praktisyns hulle heelhartige ondersteuning moet gee aan die nagraadse opknappingskursusse.

Hartlike dank word ook uitgespreek teenoor die organiseerders, die verskeie dosente, asook die Superintendent en personeel van die Nasionale Hospitaal en laastens ook die komitee met hulle baie geslaagde gholf-toernooi wat terdeë geniet is.

J. J. A. Scott, *Thaba 'Nchu*
G. H. J. de Klerk, *Bethulie*
A. F. Steyn, *Bultfontein*
P. J. du P. Vermeulen, *Lindley*
Q. Deacon, *Viljoenskroon*
S. P. Ferreira, *De Aar*

19 Mei 1958

BIBLIOGRAPHY OF MEDICAL REVIEWS

To the Editor: We would appreciate it if you would publish an announcement of the publication in June of the third annual volume of the *Bibliography of Medical Reviews.*

Review articles listed in Volume 1 and 2 were gathered as a by-product of the *Current List of Medical Literature* operation and were duplicated in the parent publication in another format. With Volume 3, however, the collection of review articles was extended to all of the current journals received by the National Library of Medicine. The result has been the inclusion in Volume 3 of approximately 600 non-*Current List* articles along with 2,300 review articles also listed in the *Current List.*

The 1958 volume of the *Bibliography of Medical Reviews* is arranged by subject with a separate author index and will contain approximately 2,900 references to review articles in clinical and experimental medicine and allied fields which have appeared largely in 1957. Copies of Volume 3 for 1958 will be available from the Superintendent of Documents, U.S. Government Printing Office, Washington 25, D.C., at a price presently estimated at $1.25.

Seymour I. Taine
National Library of Medicine *Assistant Librarian for Indexing*
Washington 25, D.C.
16 April 1958

THE IRIS OF LIFE

In a cave, denied the
Light of day, I have to live, but
Living only half in stifling gloom.
Days I sat, eyes closed,
Afraid to see the cocoon of
Darkness—that was my world. Longing for
Life and heaven, that once was mine. A
Smouldering, consuming hope; I must
Kill hope, for while it burns
This place will ever seem like
Death.
Covet not the light no longer yours,
Think not of that life so sweet.
This gloom is now your
Light, this vault is
Life . . . .
Why not forget?
When remembering brings only pain.
Life lies in the
Depths of the cave, away from the
Past.

Time brought a
Lifting of my fear. I saw
Black nothing turn to
Shadows, to objects, a
Kind of world, and I
Was not dead.
But turning once, I
glimpsed that dazzling light, an
Instant of heaven, blinding my
Hungry eyes, flooding my
Parched brain; but
So soon withdrawn.
'I cannot see! I cannot see.'
Death must be thus, it is too
Dark for life.
Yet was again my world
Emerged; but now I live in
Hope and fear
Of that which gives a
Taste of the world I had
Before, and wrecks the world that
Now is mine.

Anonymous